CHICK QUALITY: AN EVALUATION OF INNATE AND ENVIRONMENTAL FACTORS ON METABOLIC AND PHYSIOLOGICAL CHANGES IN GROWTH

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CHAPTER I

INTRODUCTION

The broiler industry is one of the fastest growing industries in animal production. The United States broiler production has increased from less than 1 billion birds in 1950 to an annual production of 8 billion broilers produced in 1998 (USDA, 2000). The poultry industry as a whole will gross 22 billion dollars in 1998 with broilers accounting for 64 % of the total (Damron, 1999). The number of chicks hatched from January to November of 1999 was approximately 50 billion chicks for broiler production (USDA, 2000). These chicks must be reared under acceptable conditions for growth and development in order to maintain this billion-dollar industry.

Numerous factors play an important role in determining chick quality and subsequent performance. A primary goal of the broiler industry is to genetically create a feed efficient fast growing bird. The nutritionists' goal in turn is to supply the resulting chicks with the nutrients to enable them to reach maximum growth and conversion efficiency. Many genetic or environmental factors may reduce chick quality and subsequently chick growth.

Chick quality begins with the hen and necessitates optimal housing and rearing. The transport of eggs and hatchery procedures also becomes very important in producing quality chicks. Sanitation, incubator temperature, air quality, transport, and holding time at the hatchery are factors that must be

carefully monitored and controlled to produce a chick of value. Once chicks are placed in the broiler house the monitoring of these birds continues for up to 7 weeks. Broiler house managers must maintain proper ventilation, water and feed quality and temperature control to optimize production success.

The studies reported herein were conducted to determine metabolic and physiological characteristics of chicks that have the best subsequent performance. It is hoped that such identification will lead to better management rearing practices that will further elevate broiler production efficiency.

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CHAPTER II

REVIEW OF LITERATURE

INTRODUCTION

Broiler production begins with genetics whereby growth and feed conversion potential are improved. Though the genetic influence plays a significant role in overall bird performance to enable birds to grow to their genetic potential chick quality must be handled and managed appropriately to minimize loss of a sellable product. Understanding metabolic and physiological homeostasis of chicks early in life is a prerequisite to producing chick with excellent quality. Only in this manner will poultry producers be able to take measures alleviating these stressors.

CHICK QUALITY

Numerous studies have been completed to create the better management systems for the hatchery and broiler house. These management systems are continually being tested to ensure continual improvements in broiler growth and feed conversion. Management practices at the hatchery are critical to chick quality and subsequent performance. The chicks produced in today's hatchery are genetically created to produce maximum profitability. However, chick stresses at the hatchery can neutralize this progress and have a detrimental effects on chick quality.

Chick quality is a combination of fertility, hatchability and management that subsequently produces a viable chick. The main objective of the hatchery is to produce a high hatchability percentage. Hatchability can be defined as the proportion of fertile chicks producing a profitable chick. Fertile eggs leave the breeder farms where they are stored at the hatchery. Embryos are placed in incubators for approximately 18 days. Embryos are then placed in hatchers for a remaining 3 days. Unusual genetic problems may cause a poor hatchability, however hatchery management can also play an enormous role in chick quality. (Etches, 1996).

Chick quality begins with the genetic selection of the chick parents. Suarez (1997) reports that genotype, age of the female breeder, and incubator environment all influence hatching performance. The age of the hen can influence variation of chick weight (Shalev, 1995), inadequate absorption of the yolk sac (Martin, 1996) and various abnormalities (Mauldin, 1996). Egg weight subsequently effects chick weight (Sinclair, 1989) can have a significant impact upon body weight gain of chicks to 3 weeks of age (Tufft, 1991). Previous studies indicated that increasing energy intake in the broiler breeder diet increased carcass protein and reduced fat in male offspring at 41 d of age (Spratt and Leeson, 1987). Economically egg weight and subsequent chick weight are related factors as a 1-gram increase of egg weight may enhance marketing weight by 2 to 13 grams (Wilson, 1991).

Early embryonic death can also begin at the breeding farm with improper collection, farm storage, transportation to the hatchery and hatchery storage of

eggs (Etches, 1996). Management factors at the hatchery are also important to ensure chick quality. Chicks can be injured, exposed to harmful disinfectants, hurt during vaccination procedures, suffocation may occur during transport or holding as well as infected with disease due to poor sanitation and/or improper holding temperatures (Keirs, 1994). Hatchery managers must follow all appropriate steps to ensure that chick quality is optimized.

Four principle factors in setting incubators including proper egg temperature, proper humidity, turning of the egg, and proper ventilation strongly influence incubator success (Wineland, 1997). Incubation temperatures must be adequate for proper growth as Hartman (1996) observed chicks incubated at cooler temperatures to have greater mortality and weight loss while being held at the hatchery. The temperature of the incubator must be controlled appropriately to ensure proper embryo growth, health and hatchability of the chicks (Mauldin, 1996). Richards (1997) reported environmental changes that take place during incubation could impact embryonic trace mineral metabolism. Hatchability will be affected by improper control of optimal incubator temperatures as well as post hatch performance and quality of the chick (Wineland, 1997).

Needlessly holding chicks in the hatcher or hatchery has created problems. Holding chicks in the hatcher for 24 h after hatch may cause a decreased immune response (Wilson, 1995) and decreased weight at time of placement (Hartman, 1995). However, holding chicks in the hatchery for 24 h, under sanitized and appropriate conditions, may help chicks to perform better on the farm by allowing them to settle after the stress of being handled and allow for

navels to heal (Vest, 1996). According the findings of Pinchasov (1993) chicks held in the hatchery for 48 h without food or water have a shortage of energy and weighed less at 14 d of age compared to chicks held 24 h. Van Der Hel (1992) also found chicks exposed to temperatures above 37 C ate less feed when measured two weeks later under normal rearing conditions.

Hatchery cleanliness is very important to reduce the spread of disease. Previous reports indicate that 75 % of samples taken from egg fragments, belting material and paper pads contained *Samonella* (Cox, 1990). Aspergillosis is a fungal infection that is 90 % hatchery related. Hatcheries provide an appropriate climate for increased infections partially due to the warmer temperatures, high moisture, overcrowding of chicks and increased presence of organic matter. (Keirs, 1994) To control the passing of parasites and diseases a proper management of sanitation in the hatchery must be established.

Improving hatchability by just one percent could have astronomical increases in net profit (Taylor, 1995). The broiler industry must work on methods to alleviate stressors, which cause poor chick quality. By understanding chick quality and subsequent growth the industry can provide therapeutic attributes to help the bird maintain growth.

EMBRYONIC GROWTH

Growth of the broiler begins in the egg as an embryo. As time surpasses the embryo grows into a vital chick. Through a series of metabolic and physiological steps the deposition of the constituents in the egg follows a normal

growth curve. Throughout the embryo's life cycle a fluctuation of chemical substances are found including protein, lipids, carbohydrates and minerals. The greatest proportion of the embryo at hatch contains protein at 56%, lipids 32%, carbohydrates 3% and minerals 9%. (Romanoff, 1967)

As the embryo grows there is an increase in total protein, lipid, carbohydrate, vitamins, minerals, enzymes and hormones. Carbohydrates including glucose begin a decline towards the end of incubation with a rise in glycogen at day 14 *in ovo,* which is indicative of liver glycogenic capabilities (Freeman, 1965). Chicks maintain a glucose concentration of 9 to 15 mM under many conditions (Langslow, 1978) including fasting (Hazelwood & Lorenz, 1959). Therefore the chicken must have an active gluconeogenic pathway to preserve blood glucose levels (Langslow, 1978). Gluconeogenesis can be defined as the pathways to convert noncarbohydrates to glucose or glycogen (Murrary, 1996). Noncarbohydrate precursors include lactate, pyruvate, gluconeogenic amino acids (aspartate, alanine, glutamate and serine), and glycerol. The gluconeogenic enzymes utilized in gluconeogenesis rise in the embryo and continue to rise until 10 - 20 days post hatch (Sturkie, 1984). *In ovo* production of blood glucose and liver glycogen increase in the final week of incubation until day 19, when the chick begins breathing at which time glycogen is mobilized (Langslow, 1978). Previous studies by Christensen (1991) indicate embryos become heavily dependent on gluconeogensis and not stored glycogen during the plateau stage (18d *in ovo*). At the plateau stage of development the embryo relies more on stored glycogen than lipid stores because more oxygen is

required to metabolize lipids than carbohydrates (Freeman, 1962). The embryo has a low concentration of plasma glucose (177 mg/dl) at 19 d *in ovo*, which may induce the enzyme glucose-6 phospatase in order for the embryo to recycle muscle lactate. Glycogen concentrations in the liver at 18d incubation averages 7.9 mg/g wet tissue rising to 13.2 mg/g wet tissue at 20 d incubation and falling at hatch to 4.4 mg/g wet tissue. (Christensen, 1995)

Proteins in the embryo rise to 63% at day 16 with a gradual decrease to 50 % at of hatching. Ammonia and urea are predominately utilized as excretory products until day 8, uric acid becomes the dominant excretory product. Protein substrates along with amino acids rise in the embryo serum until hatch. In the early stages of the embryo serum constituent is predominately lipid substrates, however, the content of protein substrates increases above the concentration of lipids at hatching. (Freeman, 1967)

The yolk contains a high percentage of lipids to serve as an energy source for embryo growth and development (Noble and Conner, 1984). Ninety to ninety-four percent of the total energy needed for the developing chick is derived from fatty acid oxidation (Linares, 1993, Noble and Cocchi, 1990). Lipid production increases with the growth of the embryo, including phospholipids and sterols (e.g. cholesterol) (Freeman, 1984). Metabolic mechanisms are said to begin around day 12 *in ovo* due to the high amounts of lipid mobilization into the embryo (Noble. et al., 1990). The yolk sac membrane has been found to contain enzymes enabling the re-synthesis of triacylglycerol. Triacylglycerol components are transported through the embryonic circulation as very low density lipoproteins

(VLDL). Activity of lipoprotein lipase (enzyme utilized for the catabolism of VLDL) has been detected in the heart at d 7, adipose tissue after day 12 and thigh muscle perhaps as an energy source from fatty acids for contraction of the heart, stored energy and growth. (Speake, 1998) The contents of liver and yolk sac membrane indicate an active cholesterol esterifying system. The embryonic liver accounts for 80 % of the total lipid present as cholesterol ester with a rapid depletion of cholesterol (oleate) at hatching. (Noble, 1984) The lipid content of whole blood remains high until hatch where concentrations are found to be 6.95mg total blood. Triglycerides were found to be in total concentration of plasma at 2.4mg. (Freeman, 1967)

The yolk sac also plays a vital role in mineral transport and metabolism. Trace minerals are transferred from the yolk sac to the embryo by the vitelline circulation to the embryonic liver where minerals are distributed to the embryo (Richards, 1997). The concentration of minerals declines from the 11^{th} day to the 21^{st} day or time of hatching with the exception of calcium, which shows an increase in concentration. There is an increase of calcium to magnesium ratio in the chick to 1.8 on day 14 to 3.6 at hatching (Freeman, 1967). It is hypothesized that this ratio increase contributes to muscle activity for the chick at pipping (Taylor, 1963 in Freeman, 1967).

Proper nutrition for the embryo begins with the hen (Speake, 1998) lipid compositions (Noble, 1984). Once the egg is laid the embryo is dependent on those nutrients deposited in the egg. Transfer, to the hatchery, handling and incubation play a role in hatchability and chick quality.

GROWTH: CHICKS TO BROILER

Broiler production is among the largest segments of the poultry industry. The goal of geneticists today is to create a bird that will grow quickly and be the most efficient in conversion of feed to be a sellable product. The nutritionists' goal is to provide the broiler a diet with a complete source of energy, protein, mineral and vitamin ration to meet the birds need for growth.

Broiler chickens are usually allowed to consume feed ad-libitum, however, there is an arising interest to restrict feed intake to minimize fat deposition. Male broilers are marketed when they have reached an approximate weight of 2.8 to 3.0 kg. Females may be reared reaching 900 to 1000 g body weight supplying Cornish hens where mixed sexes are grown reaching 1.8 to 2.0 kg body weight for use of whole birds or as cut up parts (National Research Council, 1994).

As the broiler grows deposition of fat increases, therefore it is important to provide a diet of proper amino acid balance and energy intake. A goal of the broiler producer is to maximize lean meat deposition by meeting protein needs for synthesis and minimizing fat deposition by preventing excessive energy intake. (Leeson, 1996) The quantity of fat in the carcass is effected by the dietary protein and energy ratio, however lean meat deposited is partially unaffected by change in diet due to protein in carcass controlled primarily by genetics (Leeson, 1996). Growing birds accumulate body protein at a rate of 0.6 % body weight per day. (Stevens, 1996). Protein turnover can be 5 fold higher

than the dietary nitrogen intake due to approximately 80 % of amino acids being ritualized (Swick, 1982; in Stevens, 1996). The dietary energy level selected for a diet is used as a basis for setting the other nutrient concentrations in a diet due to the idea that the bird eats to meet its energy needs as long as other essential nutrients are met (National Research Council, 1994).

The first week of life of the broiler represents 16 % of the total growth period (Gyles, 1989). Noy (1999) reported the hatched chick utilizes the yolk sac for growth of the intestinal tract even under a 48-h fast. The chick at hatch will double its weight by day 6 and increase its weight another two-fold by day 11. The feed efficiency of the chick was measured from 0 to 23 d of age and found to be most efficient at 0 – 5 d at 0.86 (g gain:g of feed). Feed conversion is best at hatch due to the utilization of energy supplied by the yolk sac. (Nitsan, 1991). As the broiler grows feed efficiency decreases (Table 2) ranging from 0.21 at 1 d to 2.01 at 56 d of age for the male and 0.22 and 2.16 for the female according to data for the Cobb-Vantress broiler.

Older broilers have a protein retention of 50 % (Buyse, 1996); yet chicks at 4 d post hatch retained less protein (30%), which may be indicative of low digestion and uptake (Noy, 1999). In an experiment by Noy (1999) it was calculated that a 45 g chick had a maintenance requirement of 4.5 kcal / d. It has been estimated that a 2000 g bird has a maintenance requirement of 120 kcal / day per metabolic body weight (Dunlop,). Understanding the dynamic pathways of energy, protein and fat is crucial to the growth and development of an efficient lean, low fat bird.

METABOLIC AND HORMONAL INFLUENCE ON GROWTH

Thermogenesis

Chicks are defined as poikilothermic (Tazawa and Rahn, 1987) where they rely on the hen or incubator to maintain their body temperature (Speake, 1998). The chick embryo is unable to regulate its body temperature at hatch until the 19th day of incubation where a rise in metabolic rate and increased thyroid activity occurs (Sturkie, 1987). Therefore chicks are described as being poikilothermic at hatch (Tazawa, 1987) and with age increasing become homethermic. Birds defined as homeotherms means that they keep a relative constant deep core body temperature (Bligh and Johnson, 1970; in Sturkie, 1986). Precocial is another term used to describe chicks that are mobile, can feed themselves, have open eyes, are covered with down (Vleck, 1987) and capable of responding appropriately to a change in ambient temperature (Sturkie, 1987). Chick body temperature gradually increases with age from 36 C at hatch to 40 C at day 24 (Dunnington, 1984). Arad (1991) found similar body temperatures for birds from hatch to 22 days of age when chicks were reared in an ambient temperature of 30 C. When chicks were reared at 35 C an increase in body temperature was seen with a range of body temperature from 38 C to 42 C. Body temperature regulation is primarily controlled by the hypothalamus which is affected by the temperature of the inflowing arterial blood (Freeman, 1984). Freeman (1984) concluded that the possible cooling of the arterial blood is achieved by blood returning from the skin of the head and the nasal cavities.

Similar studies reported by Sturkie (1986) indicate peripheral temperature receptors and temperature sensitive neurons in the central nervous system control temperature regulation in the bird.

Body temperature of the birds will appropriately respond to changes in environmental temperature by both increasing heat retention and minimizing heat loss or increasing heat dissipation. When the birds heat produced becomes stored, then the body temperature will rise and if the heat lost becomes greater than heat produced then the body temperature will decline (Sturkie, 1986). The upper or lower critical temperature can be defined as the temperature at which chicks will display detrimental effects on growth and productivity due to a change in ambient temperature. The upper critical temperature found by Van Der Hel (1990) was between 35 and 38 C. Temperatures that rise above the upper critical temperature increase heat production in conjunction with an increased body temperature (Sturkie, 1986). Berrong and Washhburn (1998) reported broilers 6 weeks old increased body temperature when exposed to high ambient temperature at 38 C compared to 21 or 32 C. Research conducted by Van Der Hel (1992) reports chicks exposed to an ambient temperature above 37 C for 48 h had a reduced feed intake, increased mortality and differences occurred in gain of body components measured after 2 weeks of feed intake and reared under normal conditions. When ambient temperature falls below the lower critical temperature birds will produce heat by shivering. Energy contributing to increased heat production, caused by shivering, is commonly derived from the oxidation of fatty acids. (Sturkie, 1986) Yahav (1997) reported broilers exposed

to 6 hours low ambient temperature (10 C) decreased body temperature from 41.1 C to 40.6 C. Broilers' form this experiment exposed to high ambient temperature increased body temperature from 41.0 C to 44.7 C.

Feed intake and subsequent growth can have an effect on temperature regulation capability of the chick (Dunnington, 1984). Teeter (1992) reported broilers increased rectal temperatures with feed intake. Previous studies conducted by Zhou (1997) reported broilers increased abdominal temperature with an increase in feed intake. Broilers exposed to high ambient temperature increased body temperature at a slower rate when food was restricted (Macleod and Hocking, 1993). It can be hypothesized that the restriction or addition of feed can influence body temperature and presumably the birds thermoneutrality.

Espira (1996) reported chicks vocally call to initiate a parent to help stabilize body temperature. It was reported that chicks which are warmed by initiating a surrogate mother to increase the ambient temperature saved 15.4 % in net energy. It is important for the chick to regulate its body temperature effectively to be able to grow properly (Dunnington, 1984). Genetics may also play a role in body temperature regulation as Berrong and Washburn (1998) reported a broiler line had a significantly higher body temperature than Athens-Canadian Randombred line. Understanding the body temperature of chicks and how chicks react to upper or lower critical temperatures is important to understanding the metabolic and physiological changes which may take place under environmental stress conditions.

Hormonal Influence on Growth

During the rapid growth increase, in post hatch chicks, plasma growth hormone is high (Harvey et. al. 1979). Freeman (1984) concluded that growth hormone in the broiler mobilizes stored lipid and reserves carbohydrate precursors for lipid synthesis. Other hormones predominate in the chicken include prolactin, insulin, glucagon and thyroxine. Prolactin holds a numerous amount of roles in the chicken including, regulation of reproduction, osmoregulation, growth and skin metabolism (Freeman, 1984). Prolactin has been found to be in high concentrations in the rapid growth phase of chicks (Harvey, 1979). Insulin and glucagon play vital roles in regulating carbohydrate metabolism. Concentrations of glucagon have been found in embyros at day 4. (Sturkie, 1986). Glucagon will stimulate glycogenolysis and lipolysis to provide energy in contrast to insulin, which stimulates synthesis of glycogen, lipids and protein (Murrary, 1996). Reported growth hormone increases the conversion of thyroxine (T_4) to triiodothyronine (T_3) in the liver of fasted or fed chicks (Kuhn, 1987, in May, 1989).

Thyroid hormones including triiodothyronine (T_3) and thyroxine (T_4) play integral parts in embryonic growth (Burke, 1990), hatchability, reproduction in turkeys (Lien, 1990), reproduction in hens (Blivaiss, 1947a, in Lien, 1990) metabolism and heat production. Specifically thyroid hormones are relate to glycogen metabolism, liver growth and heart growth in the avian embryo (Christensen, 1995). Factors that regulate the concentration of T_3 and T_4 include thyroid releasing hormone (TRH) and thyroid stimulating hormone (TSH). Thyroid hormones increase during incubation until the pipping stage at which

time T_4 will begin to decrease with a continual increase in T_3. (May, 1989) Studies show that at different stages of incubation with sex and weight of the embryo the concentration of T_4 tends to be different. (Burke, 1990)

Different stressors in which chicks are exposed can cause inhibition of the thyroid gland (Falconer and Hetzel, 1964; Falconer, 1976 in Freeman, 1984) thereby changing the concentration of T_4 and T_3 in the blood of the animal. Concentrations of T_3 and T_4 change during light and dark phases and when exposed to low or high ambient temperature. Higher temperatures will depress heat production and decrease T_3 concentration with no measurable change in T_4 (Freeman, 1984). Low ambient temperatures will increase the conversion of T_4 to T_3 (May, 1989) as well as increase oxygen consumption. Scott (1985) reported levels of T4 increased at 24 hours with chicks exposed to cold stress yet remained similar at day 7 and 14. Different concentrations of T3 and T4 have been reported when chicks were reared under lower or higher ambient temperatures. Genetic influence and stage of growth can cause differences in concentration with the influence of environmental stress.

Thyroid hormones have control over the rate of energy metabolism and the level of oxidation of all cells (Scott et. al., 1982). Newcomer and Barrett showed cardiac ventricular cells increased oxygen uptake when injected with doses of thyroxine (1960). Previous reports also show that the administration of T_4 increase oxygen consumption in myna birds (mockingbirds) (Thapliyal, 1983). Reduction in T_3 and unchanged concentrations of T_4 were found in modern strain of broilers compared to a less productive strain of broiler (Gonzales, 1999).

Modern strains of chickens are more susceptible to ascites incidence due to a higher demand of oxygen to support the increase in growth (Julian). Therefore it is hypothesized that the decrease of T3 and unchanged T4 will reduce oxygen consumption leading to hypoxia, pulmonary hypertension and ascites (Gonzales, 1999). Decuypere (1994) reported that ascites sensitive birds might have an impaired ability to respond to T_3. These changes in concentrations of T_4 and T_3 in the broiler can possibly be an innate characteristic as well as influenced by environmental conditions.

Zhou (1998) reported the influence on glucose supplementation in the drinking water of heat stressed broilers. In this report glucose supplementation increased weight gain compared to birds which received only water. Glucose supplementation also improved thermoregulatory responses in broilers indicated by a lower body temperature in chicks reared under high ambient temperature exposure. A solution of sucrose-water was found to increase body weight of chicks and improve feed conversion ratios compared to chicks receiving only water (Thaxton, 1976). Metabolic substrates measured in the blood of the bird can also indicate changes taking place under different stressors. Understanding the metabolites in the broiler under normal and stress conditions will provide a more clear picture of the metabolic and physiological changes.

Metabolic Pathways and indicators

Measuring energy, hormonal and enzymatic metabolites in blood of the chick and broiler can illustrate the metabolic changes in growth whether reared under normal or environmental stress conditions. Metabolites in the bird have

been studied extensively in broilers but literature reviewed is more sporadic in blood chemistries for younger chicks.

As the chick grows metabolic pathways become more active with a rise or fall in enzyme and substrates. The digestive organs including the pancreas, intestine and liver of the embryo play an important role in secretion and synthesis of digestible enzymes. (Nitsan et. al., 1991) A reduction in the growth of the pancreas can reduce pancreatic enzymatic hydrolysis in the intestinal lumen and subsequently cause a reduction in growth (Corring *et. al.*, 1977). The pancreas also plays a vital role in secretion of glucagon and insulin. The importance of enzymes post hatch must be active to appropriately metabolize and absorb exogenous carbohydrates and amino acids.

Carbohydrate metabolism

Carbohydrate metabolism includes the catabolism of glycogen to circulating levels of glucose and anabolism of glucose and gluconeogenic precursors to glycogen. Glycogen storage is predominately found in the liver, muscle and kidney with smaller amounts found in the heart and brain (Stevens, 1996). Liver glycogen is approximately 3.4 mg per g wet weight (Christensen, 1995) to 7.0 mg /g (Freeman, 1966) in the hatchling. Heart glycogen is found in much smaller concentrations (0.5 to 0.7 mg/g) in the hatched chick (Christensen, 1995). Realistically glycogen concentrations are found to be much higher in older birds.

Gluconeogenesis is the metabolic pathway by which glucose and other gluconeogenic precursors including alanine, glutamate, glycerol, pyruvate and

lactate are converted into glycogen. There are three regulatory, enzymatic steps in gluconeogenesis involving the conversion of pyruvate to oxaloacetate with the enzyme pyruvate carboxylase, oxaloacetate to phosphenolpyruvate with phosphenolpyruvate carboxylase, fructose 1,6 biphosphate to fructose 6-phosphate with fructose 1,6-biphosphatase and glucose 6-phosphatase to glucose with glucose 6 phosphatase. Glucokinase and hexokinase regulate the uptake and metabolism of glucose into the brain, pancreas, small intestine and liver. Gluconeogenic precursors will also follow the pathways in the cytosol to conversion of glycogen.

Glycogenolysis is the breakdown of glycogen predominately occurring in the active muscle as an energy source. Glycolysis is the metabolism of glucose into pyruvate in the cytosol. Pyruvate is transported into the mitochondria via pyruvate dehydrogenase into Acetyl CoA. Lactate is a product of glycogenolysis when the metabolism of glucose exceeds oxygen needed to convert pyruvate into acetyl CoA in the mitochondria. Lactate from the blood system will be transported to the liver to be converted into pyruvate by NAD and lactate dehydrogenase and finally to glycogen i.e. the cori cycle. Acetyl CoA will be transformed and converted to other substrates for the production of ATP in the citric acid cycle. Hydrogen ions are produced by the citric acid cycle where they are transported into energy via oxidative phosphorylation. Gluconeogenesis and Glycogenolysis are important metabolic pathways for production of energy and growth. Many steps are involved in these metabolic pathways and many stress conditions may alter these pathways.

The bird has a unique capability to maintain glucose concentrations (Table, 1) even under severe stress conditions compared to mammals (Stevens, 1996). Broilers will decrease glucose concentrations when fasted which causes a 2 – 3 fold increase in glucagon (Hazelwood, 1986b). The hormone glucagon is involved in glycogenolysis to increase the blood glucose concentrations. Glucose concentrations vary with the age, growth and energy intake. Glucose concentrations may also vary with residual yolk content as an affect of breeder age (Mahagna, 1996).

Lipid metabolism and accretion

Lipids in the bird can be made up of triglycerides, phospholipids, lipoproteins and glycolipids. The lipids present in the bird are predominately made up of triglycerides. (Table 1) Triglycerides are made up of glycerol and three fatty acids. Triglycerides are transported as lipoproteins in structures called chylomicrons. The principle deposit of triglycerides is in adipocyte cells or adipose tissue. (Stevens, 1996) Total body lipid has been reported to double every 5.5 days in the birds growth period of 1 to 6 weeks of age (Scanes, 1987). Lipid synthesis primarily takes place in the liver due to a greater capacity than the adipose tissue. There are two enzymes, which are important to lipid synthesis including acetyl CoA carboxylase and fatty acid synthase. Acetyl CoA carboxylase converts acetyl CoA into malonyl-CoA where fatty acid synthase converts malonyl –CoA into a fatty acid. Fasting causes a reduction in the rate of fatty acid synthesis, which is the opposite effect of feeding (Stevens, 1996) which will increase fatty acid sythesis.

The triglycerides in the chylomicon are catablized by lipoprotein lipase where the glycerol is carried back to the liver and the fatty acids synthesized back into triglycerides in adipose tissue or utilized as an energy source for the heart or muscle. Exogenous lipids including triglycerides are absorbed into the small intestine where they are transported as portomicrons via the portal system to the liver where they are transformed in the liver and released as part of the VLDL fraction. (Stevens, 1996).

The liver is the primary site for ketone body formation however is limited in utilizing the ketone bodies. Endogenous lipid is often transported as ketone bodies including *B*-hydroxybutyrate and acetoacetate. The synthesis of amino acids and lipids from ketone bodies increases after hatching. During a fasted state the concentration of ketone bodies especially B- hydroxybutyrate will increase in the blood of the bird. (Stevens, 1996) Concentrations of B-hydroxybutyrate (Table 1) in the plasma will also increase in birds being fed indicating lipid oxidation is occurring in the liver (Griffen, 1992; in Stevens, 1996).

Mahagna (1996) saw dramatic decreases in D(-) – B –Hydroxybutiric acid decreased on day 1 post hatch which is indicative of the transition of chicks from fat oxidation to exogenous carbohydrate metabolism.

Protein anabolism and catabolism

Proteins can be divided into two different classes, long-lived protein and short-lived protein. Long-lived proteins are degraded in the lysosomes by a group of proteolytic enzymes called cathepsins. Energy dependent pathways usually degrade short-lived proteins. Proteins will be degraded into amino acids

usually from the muscle cell when dietary protein is not present. When dietary exceeds the capacity to store protein it will be catabolized where the carbon skeletons are utilized for synthesis of fats and carbohydrates. Nitrogen is a product of protein degradation that is excreted in the form of uric acid (Table 1).

Blood proteins

Concentration of serum protein determines colloidal osmotic pressure of plasma and viscosity of the blood. The nutritional state, hepatic function, renal function and metabolic disorders of that animal influence the concentration of protein in plasma (Currie, 1995). Dietary protein also effects the serum concentration of total protein and albumin (Bailey, 1989) (Table 1). Albumin has been seen to elevate in dehydration and or shock. A decrease can indicate malnutrition and of hepatic insufficiency (Murrary, 1995). Functions of albumin include a vehicle for fatty acids, protective agent and a factor in lipid metabolism. Albumin serves as a buffer, especially in ascitic fluid. Bile, copper and nickel are also transported by albumin. Albumin also serves as antioxidant to protect damage caused by peroxyl radicals (Peters 1996).

Mineral Metabolism

Calcium and Phosphorus

One half of the calcium found in chick blood is protein-bound therefore the interpretation of calcium depends on the change in albumin and total protein (Mahagna, 1996). Calcium is important for the structure of bones but also serves components of enzymes. A deficiency of calcium and phosphorus of the hen can cause deformities in the embryo including shortened legs, bulging forehead and

protruding abdomen (Leeson, 1996). Vitamin D increases plasma calcium and phosphorus by stimulation of pump mechanisms in the intestine bone and kidney. A decrease of calcium results in an increased release of parathyroid hormone, which stimulates the kidney and eventually the intestine to increase calcium absorption. Calcitonin regulates plasma increased plasma calcium by decreasing the gut absorption and minimizing bone demineralization. Calcium and phosphorus deficiency can result in a reduction of bone mineralization. Phosphorus absorption is influenced by many factors including calcium, iron potassium and magnesium. Large intakes of Mg can inhibit the absorbtin of phosphorus by creating phytates. (McDowell, 1992).

Magnesium

Magnesium is normally absorbed through the small intestine and is inhibited by increased concentrations of calcium and phosphorus. The biggest concentration of magnesium is found in the bones with some in the muscles. Magnesium plays a vital role in the metabolism of carbohydrate and lipid metabolism as a catalyst for enzymes. However only 1 % of the total amount of body magnesium is found in blood plasma. A deficiency of magnesium can lead to impaired growth, immunity, muscle contraction and red blood cell survivability. (McDowell, 1992)

Sodium and Chlorine

Sodium (Na) makes up over 90% of the total blood cations and Chlorine (Cl) makes up two-thirds of the acidic ions in the animal. Sodium and Cl aid in the control of the passage of products in and out of the cells. Chlorine is found in

the gastric secretions including hydrocloric acid which aids in the digestion of protein. A deficeincey of Na causes a reduction in growth, soft bones and changes in cellular fluid. (McDowell, 1992) Chlorine deficeincy in chicks resulted in poor growth, high mortality, dehydration and reduced blood Cl (Leach, 1963).

Potassium

Potassium (K) is absorbed in the upper part of the small intestine. Potassium enters the cell by a concentration gradient by an active metabolic process. Sodium is the major extracellular cation where K provides 75% of the total cations within the cell. Potassium plays an important role in the acid-base balance and in transport of oxygen and carbon dioxide through the blood. A deficiency of K is unusual yet can cause cardiac and respiratory weakness (Scott, 1982).

Enzymes

Lactate dehydrogenase (LDH)

Lactate dehydrogenase is an important enzyme in the conversion of lactate to pyruvate in metabolic pathways. Lactate dehydrogenase (U/l) is defined as that amount of enzyme that converts 1 umol of lactate to 1 umol of pyruvate with the concomitatnt reduction of 1 umol of NAD to 1 umol of NADH per minute per liter of sample. Lactate dehydrogenase is intracellular therefore measurement will be indicative of cell leakage or damage. The isomer LDH_1 is released where peak values are found 35 – 43 hours after the onset of cell damage (Hermens and Witteveen, 1977).

Creatine Kinase

This enzyme catalyzes the formation of phosphocreatine which functions to generate ATP when in short supply (Voet, 1995). Total creatine kinase gives an indication of myocardial infarction. The specific isomer found in the heart, CPK-MB will be elevated within 2-4 hours after myocardial infarction and for up to 72 hours afterward, high levels can be prolonged with extension of infarct or new infarction (Harpers, 1996).

Alanine aminotransferase (ALT)

Alanine aminotransferase (ALT) (U/l) is defined as that amount of enzyme that converts 1 umole of L-alanine to 1 umole of pyruvate per minute, per liter of sample with oxidation of 1 umole of NADH according to analysis specifications of Roche. Pyruvate formed by transamination of L-alanine may then be decarboxylated to acetyl-CoA (Murrary, 1996) which yields energy through the citic acid cycle. The release of ALT in to the blood system occurs after the onset of cell damage and may be indicative of necrosis or disease (Murrary, 1996).

Aspartate aminotransferase

Aspartate aminotransferase (AST) converts aspartate to oxaloacetate which is important in the malate-aspartate shuttle to transfer of NADH across the mitochondrial membrane. Asparate aminotransferase (U/l) activity is defined as that amount of enzyme that converts 1 umole of L-aspartate to 1 umole of oxalocetate per minute per liter of sample with the oxidation of 1 umole of NADH. Aspartate is found to elevate after mycardial infarction.

Alkaline phosphatase

Alkaline phosphatase is defined as the activity which converts 1 umole of 4-nitrophenylphosphate to 1 umole of 4-nitrophenoxide ion per minute per liter of sample under the conditions specified in the assay. Alakaline phosphatase is elevated under numerous circumstances including osteoblastic bone disease and hyperparathyroidism (Murrary, 1996).

FACTORS EFFECTING GROWTH

Genetic

Modern broiler selection programs have produced fast growing feed efficient broilers. Negative indirect selection responses have at times included increase fat deposition, poor reproductive performance, leg problems and an increased susceptibility to metabolic disorders (Gonzales et. al., 1999). Scheele (1996) reported that broilers selected for low feed conversion and fast growth might effect thyroid hormone production, which may limit the bird to sustain a normal metabolic rate. Previous reports also show that birds genetically selected for feed conversion had more difficulty adapting to changes in their environment (Scheele, 1991). The genetic selection of a chick can influence its ability to grow (Scott, 1985).

Ascites

Ascites can be caused by numerous amounts of factors, which the bird encounters. One of the most significant causes of hypoxia is high altitude exposure (Cueva, 1974; Hernandez, 1987) or low atmospheric oxygen concentration caused by poor ventilation (Julian, 1987). Yahav and others

(1997) reported a significant increase in hematocrit in birds reared at 10°C versus 30°C. Feeding style can also cause the bird more susceptibility to ascites when given a pelleted feed compared to a mash feed (Shlosberg, 1992). The broiler industry has produced a fast growing feed efficient broiler. Indirect traits have surfaced including a poor respiratory system and blood circulatory system that can develop hypoxaemia (Sholsberg, 1992). Therefore, these birds placed in a cold stress environment will have an increased demand for oxygen making them more susceptible to the onset of ascites (Shlosberg, 1992).

Ascites is caused by pulmonary hypertension as the result of an oxygen insufficiency to fast growing tissues. There is a linear relationship between hematocrit and heart weight, which indicates an adaptation of the heart to increase its workload associated with changes in flow resistance (Yahav 1997). The increase in packed cell volume creates a higher blood viscosity that exacerbates pulmonary hypertension that may lead to ascites (Fedde 1996, Dewil 1996).

Chronic hypertension results in right ventricular hypertrophy and causes malfunction of the right atroventricular valve allowing blood to flow backwards into the vena cava. This process leads to liver congestion and seepage of liquid from the liver surface. When the rate of seepage is greater than the capacity of the abdominal membranes to absorb the liquid ascites develops. This eventually leads to death by respiratory failure caused by the pressure of the liquid on the air sacs. (Silversides 1997)

An increase in ascites heart index (AHI) is consistent with compensatory dilation of the right ventricle as a result of increased pulmonary arterial pressure (Hernandez, 1987). Cueva reported a possible mechanism where there was hypoxemia –induced vasoconstriction of the pulmonary arterioles and polycythemia leading to pulmonary artery hypertension, which enhances the strain on the right ventricle (1974). Ascites is measured by dividing the right ventricle by the total ventricle of the heart. The ascites heart index (AHI) can range from 0.29 without ascites to .38 with ascites as a percentage of final body weight (Beker 1995). Other findings show birds without ascites having an AHI < 25 % and those birds with ascites having an AHI 30 to 40% (Shlosberg, 1992).

Hypoxia

Hypoxia is a decrease of oxygen needed by the bird to metabolize fuels and maintain bird performance. The effects of hypoxia have been studied extensively on older birds as well as embryonic chicks. Dewil (1996) hypothesized embryos that are ascites sensitive may be predisposed to a hypoxic environment due to a longer hatching time compared to an ascites resistant line. This study reported ascites sensitive chicks had a lower heart weight, which could cause a more fatigued heart subsequently leading to a greater sensitivity to ascites. Yersin observed ascites as early as 7 days, which indicates physiological and or metabolic changes are occurring early in the bird's life (1992). Birds exposed to hypoxia conditions compensate for the lack of oxygen to tissue by increasing the contractions of the heart (Yersin, 1992). The packed cell volume as well as red blood cells and hemoglobin increase under

hypoxia conditions (Maxwell 1987, Beker 1995). Prolonged hypoxia causes the animal to try and compensate for the lack of oxygen by increasing the number of red blood cells, which increases the blood viscosity and aggravates pulmonary hypertension. Chicks reared under a high altitude environment weighed less at six weeks of age compared to chicks reared at sea level.

Hypoxia influence on metabolites

Hypoxia changes the fuels utilized by the bird to maintain homeostasis. Lactate dehydrogenase and creatine kinase are enzymes released due to myocardial damage. Maxwell (1990) and Beker (1995) indicated and increase in lactate dehydrogenase for those birds exposed to hypoxia conditions. Maxwell (1995) reported increased liver enzymes including lactate dehydrogenase and aspartate aminotransferase suggesting reduced oxygen utilization. Alkaline phosphatase was also reported in this study to decrease in ascitic birds possibly due to a bone disorder or low weight gain. Tissues which function in an anaerobic environment will also increase tissue and blood lactate (Harpers, 1996). Maxwell et al. (1986) saw an absence of liver glycogen granules in birds with ascites, which indicates gluconeogenesis is occurring from other substrates such as lactate (Diaz-Cruz 1996). Studies show birds reared under hypoxia conditions tend to decrease glucose and increase triglycerides along with substantial changes in other metabolic fuels and mineral substrates (Beker 1995).

Low ambient temperature exposure

Previous studies report detrimental effects on chicks and broilers reared under low ambient temperature (LAT) and high ambient temperature (HAT) exposure (Freeman 1967; Wiernusz 1993; Belay 1993). Cold exposure reduces body weight and increases mortality due to ascites (Benheim). Birds increase their oxygen consumption, heart rate, breathing frequency and respiratory exchange when exposed to cold temperatures (Gleeson 1985). Freeman (1965) reported day old chicks increased oxygen consumption 150% when exposed to cold stress (25°C) compared to controls (35°C). The tissues of the bird need more oxygen to metabolize substrates needed to supply heat production as a thermoregulatory response.

Birds try to regulate body temperature by increasing fuels normally utilized as energy for production are now utilizing energy as heat production. Freeman (1967) found day old chicks significantly increase ($p < 0.01$) plasma free fatty acid concentration when exposed to 60 minutes of cold stress (20°C). Freeman (1965) found 19-day-old embryos decrease liver glycogen and blood sugar ($p < 0.05$) when exposed to cold stress (20°C).

Birds increase packed cell volume or hematocrit when exposed to acclimating low ambient temperatures. As hematocrit increases the heart must work harder to pump a much more viscous blood, therefore exacerbating pulmonary hypertension which leads to the onset of ascites (Yahav, 1997).

High ambient temperature exposure

Evaporative and non-evaporative cooling are the common methods of heat dissipation. Non-evaporative cooling is the primary route broilers utilize to

dissipate heat. Birds increase surface area and shunt blood flow to the veins closest to the skin as a method of non-evaporative cooling (Bottje, 1984). Non-evaporative cooling may increase with an increased urine production congruently with increased water consumption (Belay, 1993). A continual exposure to heat stress will change the birds route of heat dissipation from non-evaporative cooling to evaporative cooling. Evaporative cooling is manipulated by increasing respiration rate (Belay, 1993).

Detrimental effects will occur to birds reared under high ambient temperature exposure. Mortality is increased when temperatures exceed the birds thermoneutral zone (Belay, 1996). Birds reared under high ambient temperature conditions reduce feed intake (Squibb, 1959) and subsequently depress growth (Belay, 1996). May (1998) reported broilers over 800 g had a better feed efficiency at a lower temperature and high ambient temperature exposure had detrimental effect on gain as weight increased. Rearing chicks under optimal climatic conditions is important for survivability and growth.

BETAINE

Betaine is a derivative of the oxidation of choline and serves many functions. Choline must be converted to betaine before methyl groups from choline can be utilized with homocystine to form methionine. Therefore dietary intake of choline and methionine directly affect the requirement of each other. (McDowell, 1989) Betaine utilized as a methyl donor can spare methionine and choline however cannot replace the two. Previous reports indicate betaine improved growth yet did not improve to the extent as choline supplementation

(Kidd, 1996). In previous studies conducted in the rat liver it has been shown that betaine is utilized to maintain methionine levels (Barak, 1982). Betaine was shown to improve growth of chicks fed a low methionine diet supplemented with betaine (Remus, 1996). Betaine has also been established in other metabolic pathways of the bird by contributing to the improvement of fatty acid oxidation by contributing labile methyl groups for the synthesis of carnitine via S-adenosyl-methionine. Carnitine helps to transport fatty acid across the mitochondrial membrane for fatty acid metabolism. This mode of action betaine takes may explain findings by Bell (1995) who reported a 12 % decrease in backfat thickness in swine fed a diet supplemented with betaine. Numerous studies have been conducted to determine the importance of betaine and not as just a by-product of choline and methionine pathways.

Numerous results have been completed indicating the efficacy of betaine with salinomycin effects on reducing the invasion of coccidiosis (Augustine, 1987; Virtanen, 1996). Coccidiosis decreases growth, feed intake and reduces the efficiency of the bird to convert feed to gain (Fox, 1987; Matthews, 1997). Coccidiosis also has detrimental effects on the intestinal tract by inhibiting the capability of the intestine to neutralize dietary insults (Fox, 1987). Betaine is reported to serve as a stabilizing agent for the structure of phospholipids, which make up the cell membrane (Rudolph, 1986). Augustine (1997) hypothesized that betaine directly improves the performance of the coccidia- infected chicks by inhibiting coccidial infection and indirectly supports the intestinal structure.

Betaine also functions as an osmotic pressure regulator, hypothesizing that betaine therefore absorbs moisture, retaining water to perhaps aid in the survivability of broilers exposed to high ambient temperature. Betaine has been defined as an osmoprotectant in *Bacillus subtilis* a gram –positive soil organism (Boch, 1994). Boch continues to explain betaines function to accumulate intracellularly thereby counterbalancing the high extracellular concentrations of osmolytes to help maintain turgor. Bagnasco (1986) also reported accumulation of betaine in the inner medulla of rabbit kidney, which also may serve to maintain intracellular osmotic balance. Further research has been conducted in Atlantic salmon where betaine tended to help these fish adapt to abrupt changes in water salinity (Virtanen, 1989). Betaines osmolyte properties needs further investigation in the broiler to determine its ability to help alleviate high ambient temperature stress.

PANTOTHENIC ACID

Pantothenic acid (PA) is a vitamin, which can be found in two enzymes, coenzyme A and acyl carrier protein. Both of these fractions are important in carrying out carbohydrate, fat and protein metabolism. (Mcdowell, 1989) It is believed that pantothenic acid, its salt and the alcohol are absorbed by diffusion in the intestinal tract (Marks, 1975 in McDowell, 1989). Researchers have not found appreciable amounts of pantothenic acid stored in animals and small quantities have been found in the blood in its free form.

Pantothenic acid as a part of coenzyme A is found in all tissues playing an important role in metabolism. Coenzyme A acts as a carrier of acetyl and other

acyl groups. Acids once bound to coenzyme A have a high potential for transfer to other groups and called "active". The coenzyme attaches to two-carbon fragments from fats, carbohydrates and some amino acids to form acetyl coenzyme A, which helps enable these fragments to enter the citric acid cycle. (McDowell, 1989).

The pantothenate derivative, acyl carrier protein (ACP) is a protein with a sulfhydryl group covalently attached to acetyl, malonyl and intermediate chain acyl groups. Acyl carrier protein plays an important role in fatty acid synthesis with the enzyme fatty acid synthetase to react with acetyl-CoA and malonyl-CoA to form Palmitate. (McDowell, 1989) Coenzyme A and ACP play functional roles in important regulatory, metabolic and anabolic pathways. Coenzyme A combines with choline to form acetylcholine, which is important for the chemical transmitter at the nerve, synapse. Coenzyme A makes succinic acid active which is the utilized in the first step of heme synthesis. (McDowell, 1989)

Requirements of pantothenic acid are variable between species and between the animals' needs for growth, reproduction and maintenance. Hens' require only 2.2 mg/kg pantothenic acid for egg production yet need 10.0 mg/kg for growth and reproduction. (McDowell, 1989) The data provided by National Research Council (1994) suggests that 10 mg/kg be provided in the diet of chicks from 0 to 8 weeks of age however the data is lacking experimentally in knowing the requirement precisely. Smith (1996) and others reviewed the requirements of pantothenic acid and determined young chicks need a minimum of 7.8 to 10 mg/kg, laying hens 10.0 to 30.0 and breeding chickens a minimum requirement

of 8.9 to 12.1 for a normal hatchability. A correlation of low dietary concentration of PA can be correlated with a decrease in coenzyme A. However liver concentrations of coenzyme A are usually maintained even under low dietary PA rations. Observations have been seen with fasting animals and the increased stimulation of the uptake of pantothenate into the liver by glucagon. (Smith, 1996) Deyhim *et al.* (1992) concluded that the recommended requirement of PA is adequate for broiler growth however an additional dietary PA would increase the nutritional value of the carcass.

Deficiencies of pantothenic acid are unusual in the chicken and can be similar to other vitamin deficiencies including biotin. The most obvious signs of pantothenic acid involve the nervous system, adrenal cortex, and skin (Scott, 1982). Pantothenic acid deficiency reduces normal egg production, hatchability with severe edema in the developing embryo. A decline in growth along with retarded feather growth and a rough plumage are noticeable signs of pantothenic acid deficiency in the chicken. Unnoticeable signs of pantothenic acid deficiency is a reduced liver concentration and discolored, hypertrophied liver. (Mcdowell, 1989).

A well balanced diet is important for utilization of all dietary substrates for optimal growth. Pantothenic acid requirements must be met for optimal growth. Further research is a necessity to determine the efficacy of pantothenic acid and its mode of action in the broiler.

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Table 1. Energy metabolites and blood hormone concentrations

	Age (days)	Sex	Glucose (mg/dl)	Triglycerides (mg/dl)	Lactate (nM/ml)	*B*-hydroxybutyrate (mg/dl)	Thyroxine (ng/ml)
Freeman (1969)	1	UK	168.8				
Pinchasov (1995	21		150-200 (p)	200-400 (p)		5.0 – 8.0 (p)	
Christensen (1995)	1		253-265 (p)				2.3-2.9 (p)
Latour (1994)	6-11		216-274 (s)	117-158 (s)			
Linares (1993)	E20					0.5 mM (p)	
Linares (1993)	1					2.0 mM (p)	
Linares (1993)	4					0.6 mM (p)	
Lien (1999)	42*		172.1 (s)	28.6 (s)			
Bowes (1988)	9	m	14.9 mmol/L (s)				
	30	m	15.7				
	9	f	14.2				
	30	f	14.9				
Meluzzi (1992)	21	both		82.6 (p)			
Brady (1977)	21-28	male			4750	362	
			208**		3040	2400	
			222***		3420	3690	

*Birds had an approximate 12 h fast before blood sample was taken.
**Birds had a 24 h fast before blood sample was taken
***Birds had a 48 h fast before blood sample was taken
(p) = Constituent taken in the plasma
(s) = Constituent taken in the plasma

Table 2. Blood proteins and excretory products

	Age (days)	Gender	Total Protein (g/dl)	Albumin (g/dl)	Uric Acid (mg/dl)	Blood, urea, nitorgen (mg/dl)
Pinchasov (1995	21		2.0 – 2.5 (p)	1.0 – 1.5 (p)	3.0 – 6.0 (p)	1.0 – 2.0
Christensen (1996)	1					
Shapiro (1997)						
Latour (1994)	6-11		3.73 (p)			
Bowes (1988)	9	m	2.56	1.19	10.9	
	30	m	3.44	1.29	6.4	
	9	f	2.68	1.18	10.3	
	30	f	3.43	1.3	5.8	
Meluzzi (1992)	21	both	3.64 (p)	1.94 (p)		
Brady (1977)	21-28	male			2.9 nmoles/ml	

(p) = Constituent taken in the plasma
(s) = Constituent taken in the plasma

Table 3. Blood mineral concentrations

	Age (d)	Gender	Calcium (mg/dl)	Phosphorus (mg/dl)	Magnesium (meq/l)	Sodium (mmol/l)	Chloride (mmol/l)	Potassium (mmol/l)
Latour (1994)	6-11		11.10 (s)	7.10-7.98 (s)				
Bowes (1988)	9	M	10.3 (s)	8.7 (s)	3.6 (s)	145 (s)	107 (s)	6.33
	30	M	10.9	9.1	2.6	154	106	4.84
	9	F	10.24	8.5	3.5	147	110	6.23
	30	F	10.8	9.6	2.7	155	108	5.08
Meluzzi (1992)	21	Both	10.45 (p)	6.1 (p)				

(p) = Constituent taken in the plasma
(s) = Constituent taken in the plasma

Table 4 Blood enzyme concentrations

	Age (d)	Sex	LDH (U/l)	Alkaline Phosphatase mg/dl	AST(U/l)[1]	Creatine kinase (U/l)	ALT[2]
Pinchasov (1995)	21		800-1200 (*p*)	4 - 8 (p)			
Bowes (1988)	9	M	801 (s)		273 (s)		
	30	M	817		184		
	9	F	886		348		
	30	F	876		182		
Meluzzi (1992)	21	Both		3562 (p)	113 (p)		

[1]AST = Aspartate Aminotransferase, [2] ALT = Alanine Aminotransferase

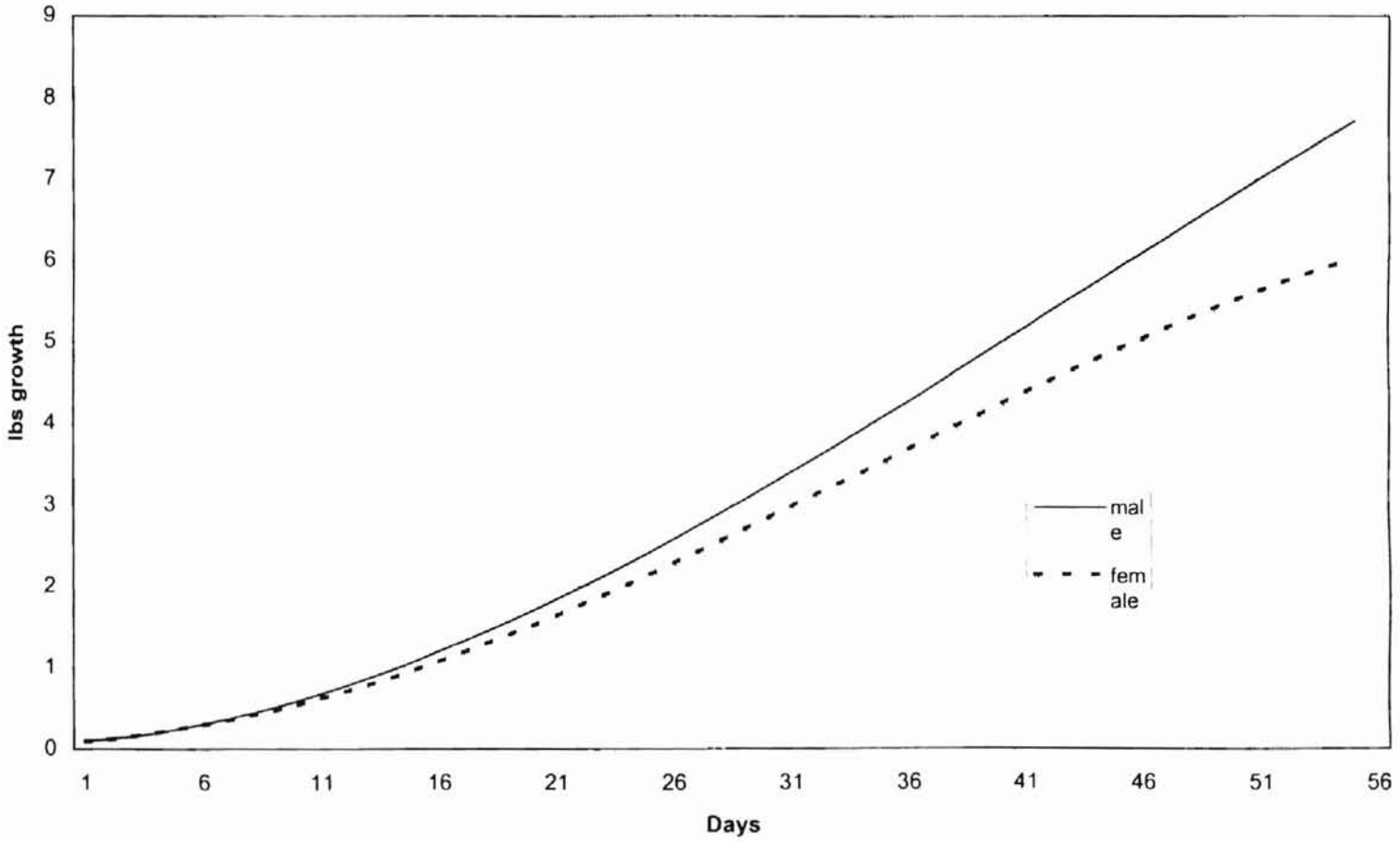

Figure 1 Cobb growth curve or male and female broilers

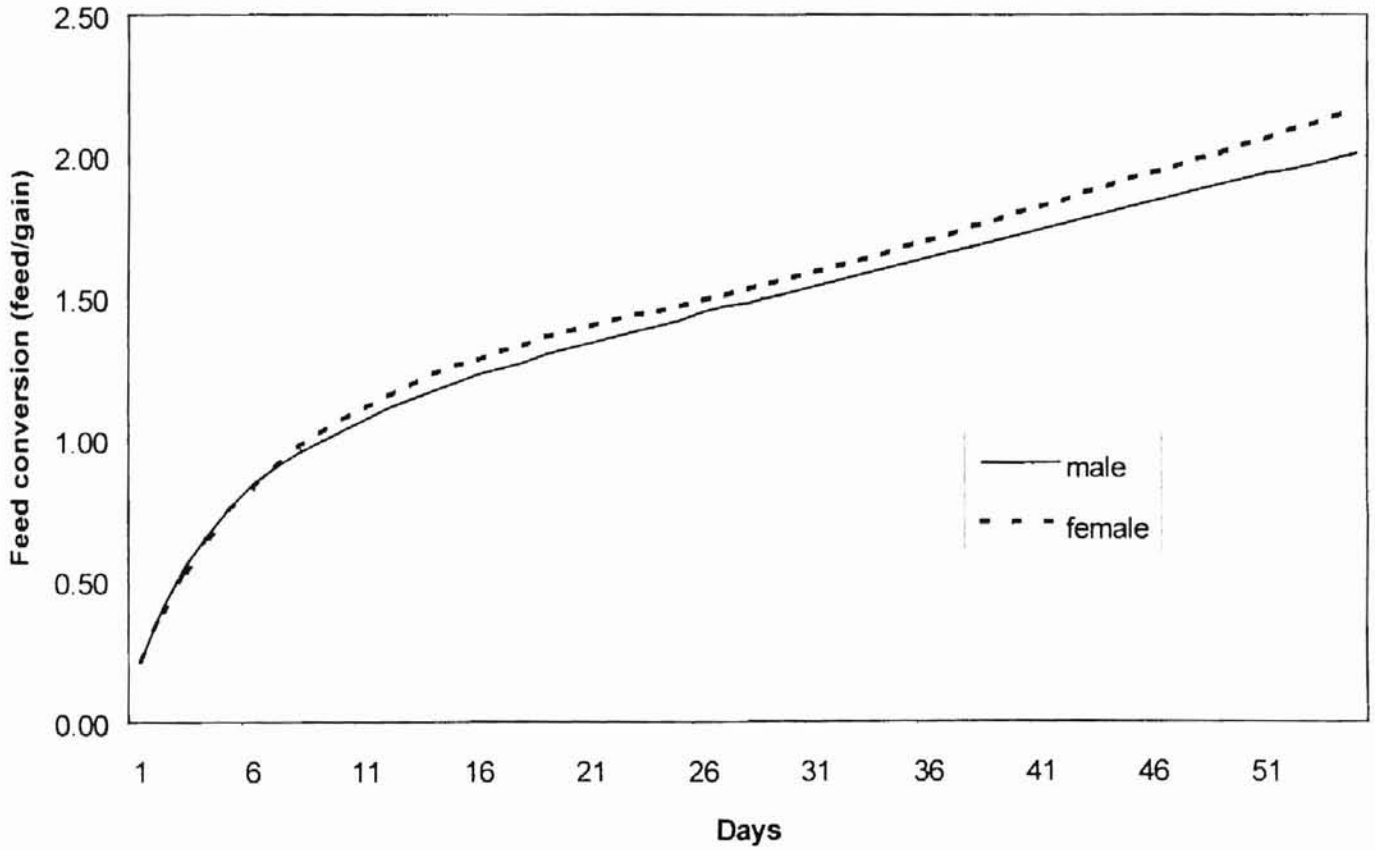

Figure 2 Cobb Feed Conversion Curve for Male and Female Broilers

CHAPTER III

AN EXAMINATION OF BETAINE ON BROILER PERFORMANCE AND THERMOTOLERANCE DURING CYCLING TEMPERTURE HEAT STRESS WITH AND WITHOUT COCCI CHALLENGE HISTORY

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ABSTRACT One study was conducted to examine betaine effects on broiler performance and thermotolerance. The experiment was partitioned into four phases. During phases 1-2 (1-21 days) chicks were allocated to floor pens and offered diets containing 0 or 0.15 % betaine with and without cocci challenge. At 23, 28, 30 and 36 days of age (phase 3) betaine supplement was switched to drinking water containing 0.1 % betaine. Also during phases 3 chicks were transferred to metabolic chambers for thermobalance assessment (heat production, HP; evaporative cooling, EC; water balance, WB; and body temperature, BT) while exposed to 24 and 35 C. Phase I feed efficiency favored betaine ($P < .01$) while other variables did not differ. Phase 2 cocci challenge

reduced all performance variables, while the betaine chicks had increased live body weight ($P < .01$) and feed consumption ($P < .05$). No interaction between betaine and cocci challenge was detected. A positive cocci exposure history, during phases 3 – 6 heat stress exposure, markedly elevated water consumption in the absenece of betaine (by 780 %) while cocci exposed chicks in the presence of betaine tended ($P < 0.1$) to have elevated water consumption (by 47 %). Birds with positive cocci history and elevated ambient temperature exposure significantly increased ($P < .01$) urine production. Betaine modulated this effect, but only when heat stress and cocci challenge was administered independently. Betaine, cocci exposure and ambient temperature significantly impacted ($P < .03$) water retention. Betaine improved water retention while ambient temperature and cocci history reduced retention. Prior to elevated temperature, birds with a cocci history had an elevated BT ($P < .03$). Upon exposure to elevated ambient temperature the birds' temperatures rose ($P < .01$). Betaine numerically reduced ($P = .14$) BT. In conclusion, betaine was noted to favorably impact both water balance and thermobalance during high ambient temperature exposure and as such warrants further study to evaluate its therapeutic properties for countering heat stress consequence.

(Key word: betaine, heat stress, thermobalance, coccidiosis)

INTRODUCTION

Broilers exposed to a heat stress environment suffer detrimental effects on growth and survivability. Previous research shows as temperature rises body weight gain decreases (Berrong, 1998) and mortality increases (Bohren, 1982).

Many researchers today are trying to determine the best environmental rearing conditions to improve broiler growth. Many studies show birds exposed to high ambient temperatures responded better to drinking water which was cool (Leeson,). Glucose supplemented in the drinking water has been shown to improve body weight gain and improve body temperature regulation when birds were reared at high ambient temperatures (Iwasaki, 1997). The broiler industry strives to improve bird survivability when exposed to high ambient temperature exposure.

The bird dissipates heat through evaporative and non-evaporative cooling (NEC). Non-evaporative cooling is the primary route of heat dissipation and can increase with an increase in urine production. Urine production has been reported to increase independently of water intake. (Belay, 1993) Evaporative cooling is the route of heat dissipation increased by respiration rate. Betaine may hold therapeutic attributes in broilers reared under high ambient temperatures by enabling the bird to reduce water intake and reduce urine production. An improved water balance enables the bird to maximize evaporative cooling efficiency thereby having a near normal respiration rate and body temperature profile. Improving respiration efficiency could reduce respiration rate and heat production that is attributed to respiration.

Many researchers have reported betaine efficacy against severe exposure to coccidiosis. Coccidia are classified as protozoan parasites that have damaging effects on the intestinal tract causing a reduction in the absorption of nutrients (Scott, 1987). Previous research defines the detrimental effects of

coccidiosis through a reduced growth, feed efficiency, and osmotic disorders of the intestine (Fox and Southern, 1987). Virtanen (1996) and Augustine (1997) reported a decrease in the severity of lesion scores of coccidia when betaine was fortified with salinomycin. Studies conducted by Matthews (1997) did not show betaine efficacy when supplemented with monensin however betaine addition of 0.1% increased average daily gain and feed intake in those chicks with a coccidiosis history.

Betaine has many characteristics to help the bird withstand stressful conditions including coccidia infection or a heat stress environment. Published data show betaine as a stabilizing agent by interacting with membrane phospholipid layers of cells (Rudolph, 1986). Ko (1994) found betaine accumulation in an osmotic and cold stressed pathogen. Accumulation of betaine in the renal medulla of the rabbit has also been cited (Rudolph, 1986). This accumulation of betaine in cells can perhaps help maintain a proper osmotic pressure and stability as Boch (1994) reported glycine betaine intracellular accumulation counterbalances high extracellular concentrations of osmolytes in *Bacillus subtilis*, a gram-positive soil organism. Studies conducted with salmon also show the influence of betaine on active transport mechanisms in the kidney and intestine (Virtanen 1989). Understanding the functions of betaine and mode of action whether a methyl donor to spare methionine and choline or as an osmoprotectant can lead to new therapeutic properties in maintaining bird health and survivability when exposed to stress conditions.

Betaines property as a methyl donor serves to spare choline and methionine in synthesis of new products. Betaine has also been proven to serve as a therapeutic advantage against invasion of coccidia. Further research in birds needs to be investigated to determine betaines role as an osmolyte in the broiler. Therefore the objectives of this study were to quantify Betaine effects on bird thermobalance (TB) (HP, EC, BT, WB), urine production and gain composition while exposed to heat stress with or without cocci challenge.

MATERIALS AND METHODS

Three hundred and twenty male Cobb-500 broiler chicks were utilized in this study. This study was completed in four phases, phase I estimated initial betaine fortification in the starter ration on performance, phase II examined betaine effects with or without cocci exposure at the beginning of the grower period. Phase III investigated betaine fortification in the drinking water with or with out cocci history under thermoneutral or cycling heat stress and phase IV examined gain composition utilizing the treatments explained for phase III.

Phase I

Chicks were placed in floor pens with 6" of clean wood shavings reared under optimal conditions till 15 days of age. Four pens per treatment with twenty chicks per pen were utilized in phase I. Feed and water were given ad libitum with and without supplemented betaine (0 % and .15 %) in the feed ration (Table 1).

Starter Phases 1 and 2 treatments:

Treatment	Betaine[1]	Coccidostat[2]
1	0	0
2	0	1
3	0.15%	0
4	0.15%	1

[1] dietary level; [2]Per Dr. Augustine recommendation for dosage level

Phases II

On day 15 chicks were inoculated with 0.5 million oocyte *Eimeria acervulina* cells in a 1ml solution per bird via the drinking water. Treatments were arranged as a 2 x 2 factorial arrangement with or without betaine (0%, 0.15%) and with or without cocci exposure. Chicks were reared until day 20 at which time 5 chicks per pen (20 chicks per treatment) were sacrificed for lesion scoring to estimate cocci severity.

Grower Phase III treatments:

Treatment	Ambient Temp	Betaine	Coccidostat
1	TN	0	0
2	TN	0	1
3	TN	0.1	0
4	TN	0.1	1
5	HS	0	0
6	HS	0	1
7	HS	0.1	0
8	HS	0.1	1

[1]Ambient temperature exposure; [2]Drinking water level; [3]Per Dr. Augustine recommendation for dosage level

Phase III

On day 21 thirty-six chicks were randomly selected and moved to calorimetric chambers for the remainder of the experiment. Treatments were arranged as a 2 x 2 x 2 factorial arrangement with 2 levels of Betaine (0%, 0.15%), 2 ambient temperatures (24 C, 35 C) with or without cocci history. The calorimetric chambers were utilized for the collection of oxygen consumption, carbon dioxide production described elsewhere (Belay, 1993). A telemetry system by Data Quest[1] was utilized for the recording of core body temperature. Chicks were implanted with temperature transmitters[2] in the body cavity of the birds' and allowed a 2-day surgery recovery and adaptation period. Birds were placed in calorimetric chambers for the collection of thermobalance variables including heat production, HP; evaporative cooling, EC water balance, WB; and body temperature, BT. Acute thermobalance data were collected during phase III on days 23 and 28 with chronic thermobalance data collection on days 30 and 36. Chicks were reared under a thermoneutral (24 C) and cycling heat stress condition. The cycling heat stress consisted of 12 h at 24 C, 3 h at 24-35 C, 6 h 35 C and 3 h 35 – 24 C (Figure 1). Chicks were fasted 12 hours prior to and during the thermobalance phase. Urine production will be collected during the thermobalance phases in mineral oil and measured by addition of known amounts of oil in the excreta collection pans.

SAS

[1] Data Sciences International 4211 Lexington Ave. North, Suite 2244 St. Paul MN 55126-6164
[2] Mini-mitter Co. Inc. 56885 Enterprise Dr. Sunriver OR 97707

RESULTS

Phase 1 Performance

Results for phase 1 represent performance data for chicks with and without betaine supplementation for a two-week period. There were no significant effects found with live body weight, feed consumption, water consumption and water to feed ratio. However, feed efficiency was improved ($P < 0.01$) from 0.786 to 0.803 for chicks consuming betaine (Table 2).

Phase II Performance with or without cocci exposure

Performance variables for phase two were measured on day 20. The broiler chicks had been exposed to a cocci challenge for a 5-day period. Cocci challenge proved to have detrimental effects on bird performance. Cocci challenge reduced ($P < 0.01$) all performance variables including live weight, feed consumption, feed efficiency, water consumption and water to feed ratio. Lesion score severity was determined by Dr. Augustine which was shown to be highly elevated ($P < 0.0001$) with cocci exposure. Betaine tended to decrease water consumption, water to feed ratio and lessen the severity of lesion score. Live weight was increased ($P < 0.01$) from 581g to 599g with betaine supplementation. A significant interaction ($P < 0.05$) was found between betaine and cocci challenge on feed consumption. Chicks without a cocci challenge with betaine fortification consumed more feed ($P < 0.05$) compared to chicks exposed to cocci challenge. (Table 3)

Phase III Thermobalance Results

Thermobalance variables including live weight gain (LWG), water consumption (WC), urine prodcution (UP) and water retained by the broiler are expressed in Table 4. Live weight gain decreased in all birds due to a 12-hour fast to reduce variability between birds. Betaine, cocci exposure, ambient temperature and the 3-way interaction impacted weight loss. Those birds that received betaine supplementation via the drinking water had an increased gain ($P < 0.05$). Birds with a cocci history had a depressed gain ($P = 0.07$) as well as birds exposed to a high ambient temperature ($P < 0.01$). Heat stress elevated body weight loss by 23%, cocci exposure elevated weight loss by 21% and betaine reduced weight loss by 19% ($P < 0.01$). A three-way interaction was found with betaine, cocci and temperature. The 3-way interaction ($P < 0.05$) was found to be the positive effect of betaine reducing the detrimental effects of heat stress or cocci history on gain.

Water consumption was effected by supplementation with betaine, cocci exposure, influence of high ambient temperatures and a betaine x cocci interaction. Birds were allowed to consume drinking water, supplemented with betaine (0.15%) throughout the thermobalance study. Water consumption increased with birds exposed to cocci history and high ambient temperature. A two – way interaction was noted between betaine and cocci challenge where cocci exposure without supplemented betaine increased water consumption 780% while, birds with coccidiosis in the presence of betaine tended ($P < 0.1$) to increase water consumption only 47%.

Urine production was markedly impacted by cocci exposure and heat stress conditions ($P < 0.001$). The betaine and cocci interaction tended to be significant (P = 0.13) where betaine numerically reduced urine production in cocci exposed chicks. Water retention was defined as the total amount of drinking water consumed minus urine production minus water loss due to evaporative cooling. The main effects of birds with a cocci history had a depressed ($P < 0.01$) water retention as did birds exposed to a high ambient temperature ($P < 0.05$). Betaine significantly improved ($P < 0.05$) water retention by 75% from the control. (Table 4)

Body temperature variables (Table 5), taken via Data Quest telemetry were taken on the birds following a 12-hour fast. Body temperature (BTI) of birds reared under thermoneutral conditions with a cocci history had an elevated body temperature ($P < 0.05$). Bird body temperature increased dramatically after exposure to the cycling heat stress. Body temperature rose from an average of 105.2 to 108.6 ($P < 0.01$) when exposed to elevated temperature. No other effects were seen however, betaine numerically lowered body temperature from 107.1 to 106.8. Betaine was also seen to numerically reduce ($P = 0.14$) the change in body temperature (DBT) rise from 1.4 to 0.99. Change in body temperature was calculated by BTO minus BTI. No other observations were seen with DBT.

Gas data (Table 6) measured within the calorimetric chambers were strongly effected by high ambient temperature. Oxygen consumption (O2C) was reduced ($P < 0.05$) when exposed to heat stress. No other effects were noted

with oxygen consumption. Carbon dioxide production was also influenced by high ambient temperature. Ambient temperature reduced ($P < 0.05$) while birds with a cocci history increased carbon dioxide production ($P < 0.10$). The respiratory quotient (RQ) was calculated by carbon dioxide divided by oxygen consumption, which is utilized to determine the substrate that is being oxidized. Main effects for cocci history, betaine supplementation and ambient temperature were all significant. All variables increased the RQ including betaine ($P < 0.05$), cocci history ($P < 0.01$) and high ambient temperature ($P < 0.05$). Evaporative cooling was estimated by the appearance of moisture or relative humidity in the outgoing chamber gases. Elevated ambient temperature and cocci exposure elevated the evaporative cooling estimate

DISCUSSION

Phase 1 failed to show betaine effects on live body weight, feed consumption, water consumption and the water to feed ratio. However, significant differences were found with betaine supplementation effects on feed efficiency. These results are similar to findings by Matthews (2000) where betaine numerically increased body weight and significantly increased feed efficiency. Lesion score was increased in birds exposed to coccidiosis that resulted in detrimental effects of coccidiosis with reduced performance variables as cited previously (Fox *et. al.*, 1987; Matthews 1997). During phase II betaine increased live body weight while cocci reduced live weight. However, no interaction was present indicating that betaine effect on weight was independent of cocci application. Remus (1996) noted betaine effect on improving growth in

45 day birds fortified with salinomycin. An interaction between cocci and betaine was noted with feed consumption where birds without cocci challenge in the presence of betaine increased feed consumption while chicks exposed to cocci had no such effect.

Betaine has been reported previously to be a protectant against osmotic stress in *Bacillus subtilis* (Boch, 1994). Betaine accumulates in the inner medulla in the rabbit (Rudolph) and Ko (1994) found betaine accumulation in an osmotic and cold stressed pathogen. This accumulation of betaine indicates its potential against osmotic stress. During the thermobalance phase of this study we found that betaine tended to improve water balance of the bird. Birds reared under heat stress increase water consumption and urine production. Birds' drinking water supplemented with betaine numerically decreased water consumption and urine production while significantly increasing water retention indicating betaine's therapeutic effect in birds reared under high ambient temperature. Heat stress effects the performance and survivability of the bird, betaine may hold properties to help alleviate these detrimental effects. Birds with a cocci history significantly increased water consumption and urine production. Cocci infected chicks supplemented with betaine tended to decrease urine production and water consumption indicating betaine is enhancing cocci infected chicks' water balance.

Birds with a cocci history had an elevated body temperature prior to heat stress exposure. This is indicative of the challenge of the birds, immune system. The high ambient temperature exposure significantly increased bird body

temperature as previously reported (Yahav, 1997; Berrong, 1998). Birds supplemented with betaine numerically reduced the severity of body temperature rise when exposed to a high ambient temperature. This trend may be indicative of an improved water balance, since thermoregulation can be effected by osmotic stress (Yahav, 1997). Ambient temperature reduced bird oxygen consumption and carbon dioxide production, which is consistent with birds slowing down their metabolic rate during heat stress exposure. Respiratory quotient increased when exposed to high ambient temperature, which is consistent with an elevated respiration rate reducing blood carbon dioxide stores. Cocci exposure and betaine elevated RQ, which is indicative of the metabolism of carbohydrate stores.

Further studies must be conducted to define betaine's true mode of action in the cocci infected and heat stressed broiler. This study shows betaine therapeutic potential in birds exposed to detrimental stress conditions. Further research with water restriction and electrolyte fortification can perhaps explain and define the potential of betaine.

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TABLE 1 Starter (0-21 days) and Grower (21-36 days) diet composition

Ingredient	Starter	Starter	Grower	Grower
Ground Yellow corn (8.8% CP)	55.2	55.2	60.1	60.1
Soybean meal (47.5% CP)	37.1	37.1	31.5	31.5
Soy bean oil	3.5	3.5	4.7	4.7
Dicalcium phosphate (DCP)	2.0	2.0	2.0	2.0
Calcium carbonate	1.2	1.2	0.9	0.9
Sodium chloride	0.4	0.4	0.33	0.33
Vitamin mix[1]	0.15	0.15	0.15	0.15
Trace mineral mix[2]	0.10	0.10	0.10	0.10
DL-methionine	0.20	0.20	0.15	0.15
Ethoxiquin	0.01	0.01	0.01	0.01
Selenium mix	0.03	0.03	0.03	0.03
Betaine	0.15	-----	0.10	----
Filler	-----	0.15	-----	0.10
Total (%)				
Calculated composition				
ME Kcal/Kg	3100	3100	3200	3200
CP (%)	23.0	23.0	20	20
Methionine (%)				
Lysine (%)				
Calcium (%)	1.0	1.0	0.91	0.91
Phosphorus (%aV)	.50	0.50	0.49	0.49
K (%)				
Na (%)	0.2	0.2	0.15	0.15
Cl (%)				

[1] Premix contained: vitamin A 720 mg, vitamin D_3 8mg, vitamin E 10.0mg/g, vitamin B_{12} 3.5 mg/g, riboflavin 2.2 mg/g, niacin 6.6mg/g, d-pantothenic acid 7.055 mg/g, cholin176.36mg/g; menadione 0.52mg/g, and biotin 44mg/g.

[2]Premix contained: manganese 12%, zinc 8%, iron 6%, copper 10%, iodine 0.1% and calcium 18%.

Table 2. Phase 1 (day15) Effects of Betaine on performance variables

	Betaine	
Variables	0%	0.15%
Live weight (g)	376	388
Feed cons. (g)	490	484
Gain/Feed	0.768[b]	0.803[a]
Water cons. (ml)	1436	1471
Feed/Water ratio	2.93	3.04

[a-b] Means within a variable with no common superscript differ significantly ($P < 0.05$)

TABLE 3. Phase 2 (Day 20) Betaine and Cocci effects on body weight (WT), feed consumption (FC), gain:feed ratio (G:F), water consumption (WC) and lesion score (LS).

Betaine	Cocci	WT	FC	LS
		(g)		
0	0	609	380	0
0	1	554	360	3.94
0.15	0	623	405	0
0.15	1	573	353	3.85
Anova	df	Probability		
B	1	0.01	NS	NS
C	1	0.0001	0.0001	0.0001
B x C	1	NS	0.05	NS

[a-b] Means within a variable with no common superscript differ significantly ($P < 0.05$)

TABLE 4. Phase III betaine (B), cocci history (C) and environment (E) effects on live weight gain (LWG), water consumption (WC), urine production (UP) and water retention (WTRETN)

		Variables							
		LWG		WC		UP		WTRETN	
Betaine	Cocci	TN	CT	TN	CT	TN	CT	TN	CT
		(g)		(ml)		(g)			
0	0	-25.39	-47.13	-1.32	11.73	18.34	23.33	-28.89	-42.79
0	1	-39.68	-46.10	24.11	65.18	37.25	67.84	-39.29	-49.81
0.15	0	-29.64	-24.72	-8.96	47.16	5.15	30.54	-26.97	-21.62
0.15	1	-29.99	-43.89	11.21	45.85	22.76	49.94	-29.43	-45.73

Anova	df	Probability			
B	1	0.05	NS	NS	0.01
C	1	0.10	0.05	0.0001	0.01
E	1	0.05	0.0001	0.001	0.05
B x C	1	NS	0.05	NS	NS
B x E	1	NS	NS	NS	NS
T x C	1	NS	NS	NS	NS
B x C x T	1	0.05	NS	NS	NS

TABLE 5 Phase III betaine (B), cocci history (C) and environment (E) effects on body temperature into (BTI), and out (BTO) of the chambers, and the change in body temperature (DBT)

		BTI[1] (F)		BTO[2] (F)		DBT[3]	
Betaine	Cocci	TN	CT	TN	CT	TN	CT
0	0	105.3	105.5	105.2	108.7	-0.05	3.05
0	1	105.5	105.8	105.5	108.8	-0.03	2.59
0.15	0	105.6	105.3	105.2	108.3	-0.57	2.72
0.15	1	105.6	105.8	105.4	108.4	-0.29	2.10
Anova	df	Probability					
Betaine	1	NS		NS		NS	
Cocci	1	0.01		NS		NS	
E	1	NS		0.0001		0.0001	
B x C[4]	1	NS		NS		NS	
B x E[5]	1	NS		NS		NS	
E x C[6]	1	NS		NS		NS	
B x C x E[7]	1	NS		NS		NS	

[1]Body temperature taken prior to placement into the chamber, [2] Body temperature taken upon removal from the chamber
[3]DBT = change in body temperature, [4]B x C = Betaine x Cocci interaction, [5]B x E Betaine x Environment interaction,
[6] E x C = environment x cocci interaction, [7] B x C x E = Betaine x Cocci X Environment interaction

TABLE 6. Phase III betaine (B), cocci history (C) and environment (E) effects on oxygen consumption (O2C), carbon dioxide production (CO2P), respiratory quotient (RQ) and evaporative cooling (EC).

		Variables							
		O2C		CO2P		RQ		EC	
Betaine	Cocci	TN	CT	TN	CT	TN	CT	TN	CT
0	0	140.3	129.2	107.1	100.0	0.76	0.78	0.75	1.94
0	1	141.4	132.7	112.1	104.3	0.79	0.79	1.13	2.22
0.15	0	139.6	128.9	108.3	102.0	0.78	0.79	0.80	2.03
0.15	1	143.7	138.2	113.3	110.7	0.79	0.80	0.92	2.10

Anova	df	Probability			
B	1	NS	NS	0.01	NS
C	1	NS	0.10	0.01	0.01
E	1	0.05	0.05	0.05	0.0001
B x C	1	NS	NS	NS	0.05
B x E	1	NS	NS	NS	NS
T x C	1	NS	NS	NS	NS
B x C x T	1	NS	NS	NS	NS

CHAPTER IV

THE EFFECTS OF PANTOTHENIC ACID LEVEL FED TO COMMERCIAL BROILERS DURING CONDITIONS PREDISPOSING CHICKS TO ASCITES.

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Pantothenic Acid Lonza

ABSTRACT Pantothenic acid is a vitamin that makes up part of coenzyme A and acyl carrier protein, which are important in lipid, protein and carbohydrate metabolism. Pantothenic acid deficiencies have been reported to have detrimental effects on growth and performance. Pantothenic acid properties were tested to define its effect on birds predisposed to ascites conditions. Pantothenic acid failed to show any effect on bird performance or decrease the incidence of ascites. The high atmospheric altitude (17.5%) had detrimental effects on bird performance and increased the incidence of ascites. The high altitude exposure decreased gain, feed consumption, feed efficiency ($P < 0.05$) and increased hematocrit and ascites heart index ($P< 0.01$).

INTRODUCTION

Pantothenic acid is found in two enzymes, coenzyme A and acyl carrier protein (ACP) which are involved in many reactions in carbohydrate, fat and protein metabolism (McDowell, 1989). Research experiments are variable in the

amount of pantothenic acid needed to meet the bird's requirement. The National Research Council (1994) for poultry recommends 10 ppm in broilers 0 to 8 weeks of age.

Ascites caused by a lack of oxygen needed to supply fast growing tissues, increases packed cell volume causing right hypertrophy of the heart and pulmonary hypertension that leads to edema or more commonly called "water belly". Beagle and Begin (1976) reported that diets marginally deficient in Pantothenic acid increased ration heat increment along with a reduced growth rate and reduced feed efficiency. If the birds heat increment is increased the need for oxygen increases thereby depleting oxygen availability especially if birds are reared in an environment predisposing to ascites. The objective of this study was to determine pantothenic dietary levels and therapeutic effects in birds reared under conditions predisposing to ascites.

MATERIALS AND METHODS

Two hundred and fifty-six male Cobb-500 broiler chicks were utilized in this study. Treatments were arranged as a 2 x 4 factorial arrangement with two atmospheric oxygen concentrations (17.5 % vs. 20.6%) and four dietary treatments. Birds were allocated within 40 respiratory chambers described elsewhere (Belay, 1993) and reared under mild cold stress environment (31°C) for a 2 week feeding period. Starter ration composition was representative of commercial formulation and was calculated to 3,159 kcal ME_n/kg and 23.47% CP. The starter ration was fed ad libitum as a mash diet with 7.5 mg/kg pantothenic acid in the diet.

Statistical Analysis

Data were analyzed using the general linear models (GLM) procedure of SAS (SAS Institue, 1990). Difference among treatments were identified with least significant differences at $P < 0.05$.

RESULTS

There were no interactions detected between altitude and oxygen level. Therefore main effects were analyzed for Pantothenic Acid and atmospheric oxygen (Table 1). There were no effects of pantothenic acid level on any variables measured. Birds exposed to high atmospheric oxygen caused detrimental effects on bird performance. Birds reared at 17.5% oxygen consumed less feed, reduced gain, live weight, feed efficiency and increased the water to feed ratio ($P < 0.05$). The birds reared under a high altitude had an increased incidence of ascites heart index ($P < 0.01$) and hematocrit ($P < 0.01$).

DISCUSSION

Pantothenic acid deficiency in the bird can decrease gain and feed efficiency (Smith, 1996), increase dermatitis (Raidal, 1995), and increase the heat increment of feed (Beagle, 1976) possibly increasing the susceptibility of ascites. In this study Pantothenic acid did not show to have efficacy on reducing the onset of ascites. Birds reared under conditions predisposing to ascites had increased AHI and hematocrit as reported numerously (Sillau, 1980; Maxwell 1987; Beker, 1995).

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TABLE 1. Pantothenic Acid and Atmospheric Oxygen Effects on Performance and Ascites Incidence

Variables	Treatments (ppm)					Oxygen Concentration (%)		
	7.5	10.0	20.0	40.0	Probability	17.5	20.6	Probability
Feed cons.[1](g)	415	409	412	415	0.68	392	434	0.01
Gain/feed	0.86	0.87	0.88	0.86	0.54	0.85	0.88	0.01
Adj. Gain/feed[2]	0.96	0.98	0.98	0.97	0.40	0.96	0.98	0.02
Gain (g)	358	356	361	359	0.81	334	383	0.01
14d weight(g)	401	399	403	402	0.90	376	426	0.01
Water/fd ratio[3]	2.64	2.67	2.58	2.64	0.41	2.70	2.56	0.01
Hematocrit(%)	33	32	32	33	0.59	35	30	0.01
Ascites index	0.20	0.19	0.20	0.19	0.77	0.21	0.18	0.01
Survivability(%)	0.98	0.97	0.95	1.00	0.64	0.97	0.98	0.62

[1]Feed consumption, [2]Feed efficiency adjusted for birds that died, [3]Water to feed ratio

CHAPTER V

EVALUATION OF BODY COMPOSITION THROUGH PROXIMATE ANALYSIS TO EXAMINE THE EFFICACY OF UTILIZING X-RAY DENSITOMETRY

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ABSTRACT Two experiments were conducted to determine body composition changes overtime with or without exposure to hypoxia (17.5, 20.6). Experiment one was conducted to determine changes in body and tissue water balance over a two-week period. Chicks exposed to Experiment two was conducted to examine suitability of the "Whole Body Bone Densitometer" (Hologic QDR™ – 1000/w)[1] system for estimating body composition. Values were compared with values obtained by proximate analysis, whereby, protein was estimated as nitrogen x 6.25, lipid as ether extract and total minerals as residual ash. Positive relationships were noted between proximate analysis values for nitrogen, ether extract (EE) and ash with densitometer values for lean, fat and skeletal mass (bmc). However, regression equations were needed to precisely relate results. The "best fit" equations, used to convert densitometer values to those attained via proximate analysis were as follows: 1) protein = 13.55 + lean * 0.156 [R =

0.83, P < 0.01]; 2) EE = 10.90 + fat * 0.3395 [R = 0.59, P < 0.01]; 3) ash = 4.58 + bmc * 0.966 [R = 0.75, P < 0.01]. Results suggest that the "Whole Body Bone Densitometer" offers the opportunity to collect body composition on live anethesized birds that may be effectively related to proximate analysis values. (*Key words*: body composition, proximate analysis, x-ray densitometer, carcass fat, carcass protein)

INTRODUCTION

There are many procedures in estimating body composition of the chicken. Many of these are laborious and costly to accomplish. Wang (1992) suggested five levels of body composition models including atomic, molecular, cellular, tissue-system and whole body. Birds utilized to determine whole body composition are often sacrificed making it difficult for the researcher to follow the true composition of a single bird throughout its' growth period. A review conducted by Foegelom (1997) suggested that there is a high variability between body composition methodologies due to different instruments and calculations. Therefore it is imperative to continue researching different potential procedures to determine body composition of the bird without sacrifice in order to follow a truer estimation of fat, protein, and energy deposition. The objective of this study was to determine the efficacy and accuracy of utilizing the Hologic x-ray densitometer with chicks exposed to a hypoxic environment.

MATERIALS AND METHODS

Experiment 1 was conducted to determine the change in total body and tissue dry matter overtime in chicks exposed to two atmospheric oxygen

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[1] Hologic. Waltham, MA 02154

concentrations (17.5%, 20.6%). A high atmospheric oxygen has been reported numerous times to reduce body weight, feed efficiency and have detrimental effects on physiological (Maxwell, 1995) and metabolic processes. The oxygen concentration in the area this study was conducted is defined to be 20.6% oxygen therefore this was utilized as the control oxygen concentration. Eighty Cobb-500 broiler chicks were randomly distributed into in 8 calorimetric chambers described elsewhere (Belay, 1993), and reared until 14 d of age. Fifteen chicks were processed to define initial body and tissue dry matter. Chicks were given a starter mash diet ad libitum with MJ/kcal and %CP. Hematocrit or packed cell volume was taken on all birds prior to processing. The right breast tissue was taken, weighed and placed in 100 C drying oven. The remaining carcass was placed in pans and dried to estimate dry matter.

Experiment two was conducted to determine carcass composition through proximate analysis to determine the efficacy of x-ray densitometry. Twenty-four birds were randomly placed in calorimetric chambers with two atmospheric oxygen concentrations as described previously. Birds were reared until 14 days of age with food and water given ad libitum. Ten birds were initially sacrificed on day 0 to estimate initial body composition. Hematocrit was taken, birds were expended, scanned by x-ray densitometry and analyzed by proximate analysis procedures. Values collected from x-ray densitometry were compared with values obtained by proximate analysis, whereby, protein was estimated as nitrogen x 6.25, lipid as ether extract and total minerals as residual ash.

Statistical Analysis

Data were analyzed using the general linear models (GLM) procedure of SAS (SAS Institue, 1990). Difference among treatments were identified with least significant differences at $P < 0.05$.

RESULTS AND DISCUSSION

Experiment 1 results

Variables measured in experiment 1 included weight, hematocrit (HMT), ascites heart index (AHI), tissue dry matter (TDM) and bird dry matter (BDDM) (Table 1). The interaction between oxygen and age was found to be significant with HMT ($P < 0.001$) and AHI ($P < 0.05$). Live weight (LWT) ($P < 0.05$) tissue dry matter ($P < 0.01$) and BDDM ($P < 0.05$) increased overtime with age. Live weight increased from 38g at hatch to 419g at day 14. Tissue dry matter increased from 8.1% at hatch to 35.7% at day 14. Bird dry matter also increased from day 0 at 23.7% to 26.9%. Romanoff (1967) reported that the chick embryo decreases in total water from day 3 at 92% to the day at hatch, 79%. Romanoff suggests this increase overtime may be due to an increase in kidney function. Christensen (1995) found total body water to average 68% in hatched chicks. The percent water found in the present study decreased from 76.0% at hatch to 73.0% at day 14. Hematocrit increased overtime with or with out exposure to hypoxic conditions which could be a function of the percent body water decreasing overtime.

Experiment 2 results

Hematocrit increased overtime ($P < 0.01$) with no differences found in AHI or any serum chemistries measured exposed to 17.5% oxygen (Table, 2). Beker (1995) found differences in serum chemistry concentrations when birds were exposed to an oxygen concentration of 13.0%. Body composition variables measured by proximate analysis procedures showed a decrease in fat ($P < 0.05$) and protein ($P < 0.01$) percent on a dry matter basis (Table 3). Fat (24.0%) was found to be similar in control birds with previous reports by Latour (1994) with body fat at 21.0% at 11 d of age. This study concluded a protein percent of 64% where Latour (1994) reported protein to be higher at 84.6%. Van Der Hel (1992) found protein to 52.8g, which would be closer to the protein levels we estimated.

Protein, ash and fat estimated for x-ray densitometry were regressed against protein (Figure 1), fat (Figure 2) and ash (Figure 3) estimated by proximate analysis to determine the efficacy of utilizing the x-ray densitometer as a means of following body composition changes overtime. Significant regression equations were found when predicting body composition. Mitchell (1997) reported efficacy of x-ray absorptiometry in prediction of fat in broilers weighing over 2000g, however it was indicated that the size of the bird and program utilized to estimate composition tends to be variable. Regression equations utilized on day 0 for birds scanned and processed were not significant. This could be due to the soft tissue and cartilage differences in small chicks. Therefore we conclude the x-ray densitometer has potential to be utilized as a

way to determine body composition in older broilers utilizing appropriate regression equations.

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TABLE 2 Experiment 2 Performance variables (d 14) exposed to two altitudes

	Atmospheric Oxygen		
Variables	17.0 %	20.6 %	Probability
Hematocrit	35.41	29.96	0.0001
Ascites Heart Index	0.19	0.22	NS
Glucose (mg/dl)	190.73	221.90	NS
Total Protein (g/dl)	2.65	2.57	NS
Albumin (g/dl)	1.11	1.02	NS
Sodium (mmol/dl)	154.48	152.04	NS
Magnesium (meq/l)	2.38	1.98	NS
Calcium (mg/dl)	6.12	7.01	0.10
Phosphorus (mg/dl)	6.95	6.89	NS
Potassium (mmol/l)	6.09	5.02	NS

TABLE 3. Experiment 2 Atmospheric oxygen effects on body composition (d 14) variables determined by proximate analysis.

	Atmospheric Oxygen		
Proximate Analysis	17.0%	20.6%	Probability
Fat (g)	23.97	28.39	0.01
Ash (g)	10.16	11.03	NS
Protein (g)	66.96	71.96	0.05
X-ray densitometer			
Fat (g)	24.76	26.56	NS
Ash (g)	10.32	10.66	NS
Protein (g)	67.15	71.05	0.05

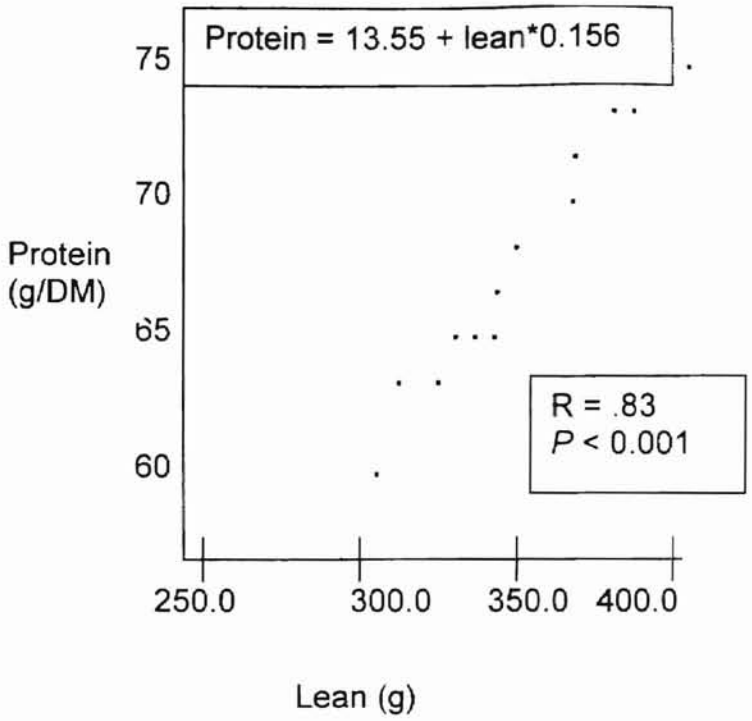

Figure 1. Lean estimated by x-ray densitometry regressed against protein calculated by proximate analysis

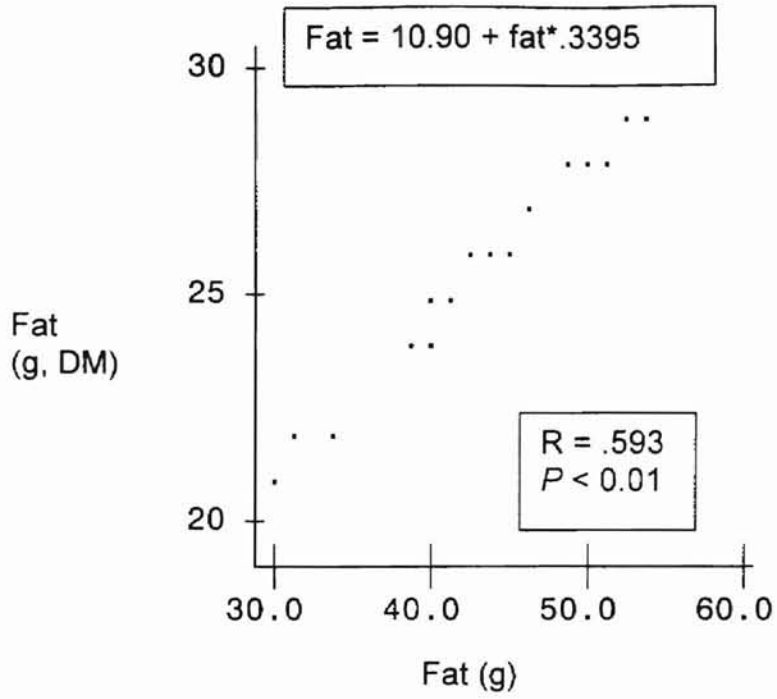

Figure 2. Fat esimtated by x-ray densitometry regressed against fat by proximate analysis

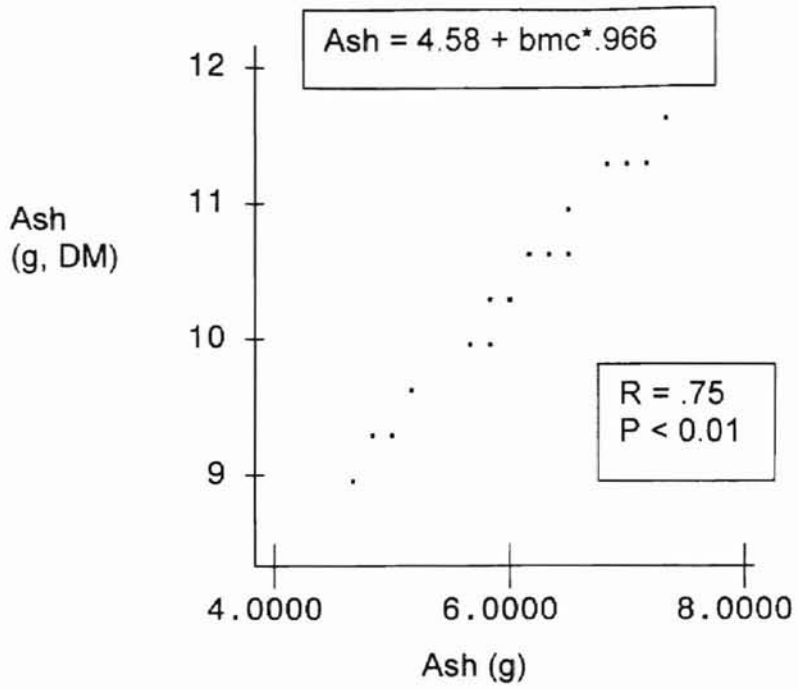

Figure 3. Bone mineral content estimated by x-ray densitometry regressed against ash calculated by proximate analysis

CHAPTER VI

INFLUENCE OF AMBIENT TEMPERATURE AND INITIAL CHICK BODY TEMPERATURE ON SERUM AND TISSUE CHEMISTRIES.

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ABSTRACT Previous studies conducted in our laboratory suggests that initial chick body temperature (at hatch) is positively correlated with live weight gain, gain/feed ratio and ascites resistance. In this regard three experiments were conducted to evaluate effects of body temperature at hatching (HBT), on subsequent broiler performance under varying combinations of feeding and ambient temperature (30, 24 C). Chicks were initially divided into two groups according to falling above (HT) or below (LT) mean hatching body temperature. Traits measured throughout the study included body temperature (BT), body weight, liver glycogen, and 18 serum analytes. During experiment 1, chicks fasted to 24 and 48 h at 24 C, exhibited increased ($P < 0.05$) body temperature whether falling above (HT) or below (LT) mean body temperature (101.9 F). Chicks exhibiting a high body temperature increased ($P < 0.01$) BT at a greater rate than low body temperature chicks. Liver glycogen, blood urea nitrogen, uric acid, total protein and albumin increased whereas glucose, triglycerides, lactate, and *B*-hydoxybutyrate declined ($P < 0.05$) with time post hatch. Experiment 2

was conducted to evaluate body temperature change in fasted chicks housed at 30 or 24 C. Chicks reared under low ambient temperature (LAT; 30 C) reduced ($P < 0.01$) BT and produced less ($P < 0.01$) carbon dioxide regardless of body temperature class. Experiment 3 was conducted to determine hatching temperature in fed chicks housed at two ambient temperatures (30, 24 C). Body temperature for HT chicks increased ($P < 0.01$) markedly compared to LT chicks. A positive correlation was noted between BT and subsequent feed consumption ($R = 0.70$, $P < 0.05$) as well as BT and live gain ($R = 0.91$, $P < 0.01$) at 30 C but not 32 C. Data suggest that chick performance and metabolism are impacted by initial BT at hatching. Whether these effects are the result of innate chick characteristics and/or the hatch environment remains unknown.
(*Key words*: broiler, metabolism, chick quality, body temperature, ambient temperature)

INTRODUCTION

Among genetic influences impacting bird stress resistance and overall performance may be genetic programming for metabolic set point. Set point for body temperature defined as the point at which birds reach a stable body temperature with maximum growth. Animals are presumed to have a metabolic set point that regulates body temperature. Body temperature is a balance between heat production, dependent upon oxygen consumption, and heat dissipation. In other words an increase in chick body temperature results partly by an increase in metabolically active mass and metabolic rate (Sturkie, 1986). It is documented that initial chick body temperature is not constant and is indeed

evolving to a reported norm of 41 C (Freeman 1984). An experiment conducted by Hocking (1985) showed heavy meat type stocks had a reduced ($P < 0.01$) BT compared to Leghorn or medium sized meat type chicks. Whether birds with a high or low body temperature have superior performance, or have a set point that effect ability to cope with stress is not known.

Differences in the newly hatched chick set point for body temperature may be the result of specific genetic effects or metabolism, or the availability of substrate to sustain metabolism. Previous studies hypothesize that chicks with a higher body temperature have better feed conversion compared to chicks having a low body temperature (Teeter and Skinner-Noble, unpublished). This innate factor could indicate differences in metabolic energy store and utilization subsequently effecting chick quality, performance, and survivability when exposed to environmental stress factors.

Chick performance is impacted by genetics (Christensen, 1995) as well as environmental factors (Freeman, 1966). Developments in accordance with blood chemistries have been effectively measured in embryonic and adult broilers. The egg is rich in lipids and proteins providing energy for chick survivability (Romanoff, 1967, Speake, 1998). Studies conducted have determined the metabolic pathways in which chick embryos and hatched chicks utilize energy stores (Rinaudo, 1976; Langslow, 1978, Watford, 1981). Chicks from different genetic lines may differ in fuel reserves as Christensen (1995) found with liver and heart glycogen and glucose concentrations.

Chicks will respond to stress conditions metabolically and physiologically. As a thermoregulatory response the bird will increase its oxygen consumption (Gleeson, 1985) to metabolize substrates (Freeman, 1966; Freeman, 1967) when exposed to cold stress conditions. Pervious studies conclude day old chicks increase their oxygen consumption 150% when exposed to severe cold stress (25 C) (Freeman, 1965).

The objective of the study described herein is to determine if chicks differing in body temperature set point also differ in growth rate, liver glycogen and serum chemistries.

MATERIAL AND METHODS

Initial Bird Selection, Categorization and Processing

Three hundred and sixty Cobb X Cobb chicks were randomly extracted from the day's hatch and categorized by body temperature. Chick body temperature was taken by way of the cloaca utilizing a DeltaTRAK[1] digital temperature probe. Body temperature was classified as high (HT) or low (LT), based on the mean body temperature of the first 25 chicks. Following initial categorization and processing, birds were transported to calorimetric chambers described elsewhere (Belay, 1993). Birds utilized in the following three studies were extracted at random from these two populations. Chicks were fed a complete starter ration with metabolizable energy (kcal/kg) and 23% crude protein (Table 1).

[1] DeltaTRAK, Pleasanton, CA 94566.

Tissue and Blood Analysis

Blood was collected on all chicks by cardiac puncture. Blood was collected in Corvac[2] sterile tubes and allowed clotting for twenty-four hours. Blood was then centrifuged at 3000 rpm for 25 minutes. Serum was removed and frozen in polyethylene tubes until subsequent analysis. Blood was collected for lactate separately and treated with trichloracetic acid according to specifications of Roche. Blood serum or lactate supernatant was analyzed utilizing a Cobas Mira[3] wet chemistry analyzer. Serum samples collected were analyzed for sodium, potassium, calcium, magnesium, chloride, phosphorus, glucose, triglycerides, total protein, albumin, uric acid, blood urea nitrogen, creatinine, lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase and creatine kinase. Roche kits[4] utilized in determining serum concentrations include calcium (No. 44033), magnesium (No. 44169), chloride (No. 44029), phosphorus (No. 44031), glucose (No. 47382), triglycerides (No. 44120), total protein (No. 44903), albumin (42332), uric acid (No. 47273), blood urea nitrogen (No. 47380), creatinine (No. 47003), lactate dehydrogenase (No. 47259), aspartate aminotransferase (No. 42381), alanine aminotransferase (42375), alkaline phosphatase (44553), creatine kinase (44402). Sodium and potassium values were assayed via sodium and potassium selective electrode module (No. 46997, 46998). Sigma reagents were utilized in measuring lactate (9826-A) and B-hydroxybutyrate (310-A).

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[2] Corvac
[3] Roche Diagnostics Systems Inc., Montclair, NJ 07042-5199.
[4] Hoffman-LaRoche, Nutley, NJ 07042.

Liver tissues were collected from the chicks immediately after blood collection and frozen in liquid nitrogen. Livers were stored in whirl pack bags or wrapped in aluminum foil in a –80 freezer. Analyses of liver for presence of glycogen were completed according to methods of Dreiling (1987).

Data were analyzed utilizing General Linear Models of SAS (1990) for all variables measured.

Experiment One

The first experiment was conducted to examine the changes in initial body temperature and metabolic substrates overtime. Treatments were arranged as a 2 x 3 factorial design with two body temperature classes, (HT vs. LT) and three processing times (0, 24 and 48 post hatch). Chicks were deprived of food and water and kept in transport boxes, housed at 32 C to represent a shipping environment. Variables collected during processing include liver glycogen plus serum and liver dry matter plus serum. Hour represents hours old and/or the hour at which time the chicks were removed from the hatchers.

Hour 0 post hatch processing was completed at the hatchery before transport. To determine if change occurs in liver glycogen due to handling or heart puncture, liver glycogen and dry matter was collected from chicks without collection of blood at hour 0. Chicks were transported from the hatchery to the OSU avian climatological research laboratory where all remaining processing days took place. Body temperature and weight temperature was taken prior to serum, liver glycogen and dry matter collection at hours 24 and 48. Sixty chicks were processed representing thirty high and thirty low body temperature classes.

Experiment Two

The second experiment was conducted to determine if body temperature plays a role in oxygen utilization in fasted chicks. Treatments were arranged in a 2 X 2 factorial design with two body temperature classes (HT vs. LT) and two ambient temperatures (30.0 C vs. 32.0 C). Twenty-four chicks were randomly placed into metabolic chambers described elsewhere (Belay and Teeter, 1993). Chambers were allocated into two rooms each chamber representing a body temperature class. Chicks were placed into metabolic chambers at hour 24 and processed at hour 56.

Experiment Three

Experiment three was conducted to determine the effects of body temperature on oxygen utilization in fed chicks. Treatments were arranged in a 2 x 2 factorial arrangement with two BT classes (HT vs. LT) and two ambient temperatures (30 C vs. 32 C). Twenty-four chicks were randomly placed into individual chambers allocated into two rooms. Chicks were fed a mash starter ration given ad libitum throughout the remainder of the trial. Chicks were placed into metabolic chambers at hour 60 and removed at hour 168. Body temperature and weight was taken prior to placement and after removal from the chambers.

RESULTS

The 360 chicks, collected at the hatchery, ranged in weight from 32.3g to 53.6g with an average weight of 43.7g. Body temperature for these chicks ranged from 98.0 F to 104.0 F. Chicks classified as HT (150 birds) ranged in body temperature from 102.2 F to 104.0 F with a mean body temperature of

102.7 F. The weight of the HT body temperature chicks ranged from 34.6g to 50.7g with a mean of 43.4g, while chicks classified as LT (204 birds) ranged form 32.3g to 53.6g with an average weight of 43.9g. Body temperatures of the LT group ranged from 98.4 F to 102.0 F with a mean of 101.4 F. Processing at the hatchery is representative of blood chemistries from chicks at 0 hours following removal from the hatchers (Table 2).

Experiment 1 results

Results of experiment 1 reveal a significant interaction between body temperature class and time body temperature was taken at time of processing. Body temperature recorded for chicks at hours 0, 24 and 48,increased ($p < 0.05$) with time post hatch (Figure 1) independent of body temperature class. Change in chick body temperature, calculated as body temperature of chicks at hatching from BT at time of processing. Chicks categorized as LT ($P < 0.05$) increased their body temperature at a greater rate compared to HT chicks (Table 3, Figure 2). Weight was depressed ($P < 0.01$) as the hours birds were fasted increased. The change in weight was calculated by subtracting individual weight by the mean weight of each BT class taken at the hatchery. The change in weight also decreased as time post hatch increased ($P < 0.01$) (Table 3).

The interaction of high and low body temperature class and hour was only significant with glucose therefore all other variables were expressed by mean values for main effect of time. Chicks with a low body temperature (LT) significantly decreased serum glucose concentrations at all hours. High body temperature (HT) chicks decreased glucose concentration yet leveled off at hour

48 (Table 4, Figure 3). Total liver glycogen, blood, urea and nitrogen, uric acid, total protein and albumin increased while glucose, triglycerides, lactate, and *B*-hydoxybutyrate significantly decreased overtime ($P < 0.05$) (Table 5). Chicks with a HT had a higher serum uric acid (11.49 mg/dl) concentration than LT chicks (8.24 mg/dl) which indicates chicks with a higher body temperature where utilizing amino acids to a greater extent.

Minerals that were quantified either numerically or significantly increased hour 0 to hour 24 and tended to level off at hour 48 (Table 5). Enzymes measured in the serum are indicative of cells damaged not as enzymatic response to substrates. Enzymes including lactate dehydrogenase, alanine aminotransferase, aspartate aminotransferase and creatine kinase increased overtime from hour 0. Changes in the enzymes measured tended to be sporadic.

Experiment 2 results

Chicks classified as having a high body temperature had a higher body temperature than those chicks classified as having a low body temperature ($P < 0.01$) prior to placement into the metabolic chambers (Table 6). Chicks exposed to a twenty-four hour low ambient temperature significantly decreased their body temperature ($P < 0.0001$) whether having a HT or LT (Table 7). Carbon dioxide production was positively correlated with body temperature taken once removed from the metabolic chambers ($R^2= 0.64$, $P < 0.01$). Chicks exposed to a low ambient temperature (30 C) produced less carbon dioxide whether having a HT or LT ($P < 0.01$) (Figure 4). There were no differences seen with carbohydrate stores in chicks exposed to a mild cold stress, however, chicks designated as

having a LT had a significantly higher concentration of total glycogen in the liver (4.73g) compared to those chicks with a HT (2.52g) ($P < 0.05$). There were no other effects of cold stress or body temperature class on energy, mineral or enzyme substrates.

Experiment 3 results

There were no significant differences found within experiment three except for body temperature variables (Table 8). The change in body temperature of the LT chicks increased dramatically compared to HT chicks ($P < 0.01$). A positive correlation was apparent between body temperature taken once removed from the chambers and feed consumption of those chicks (0.74, $P < 0.05$) which indicates chicks with a high body temperature are eating more feed. Increased feed consumption will possibly increase the metabolic rate subsequently causing an increased body temperature of that bird.

DISCUSSION

The weights of chicks at hatch were close to the expected values of the Cobb 500 growth curve. Myhre (1977) reported colonic body temperature in bantam chicks to range from 101.3 to 105.8. Body temperature increased until they reached adult body temperature (105.1) at 9 d of age. Chicks classified as having a lower body temperature at hatch significantly increased body temperature at a greater extent than high body temperature chicks, this trend was seen throughout all experiments. Myhre (1975) attributed the increase in body temperature of chicks to be a controlled condition where the set point is low at hatch and rises to adult temperature during the first week. Body temperature

maintenance incurs at an energetic cost as stated by Brown (1999). A study was conducted on ostrich chicks where energy expenditure was reduced to half from 25 C to 35 C in day old chicks (Brown 1999, unpublished). Previous reports indicate high ambient temperature exposure during the first two days post hatch reduces feed intake and growth within the next 2-wk growth period (Henken, 1987; in Van Der Hel, 1990). As reported in this study lower ambient temperature reduced body temperature. However we found oxygen consumption decreased with an increase in carbon dioxide production in chicks exposed to a 24-h mild cold stress. Our hypothesis is chicks exposed to a lower ambient temperature within a twenty-four hour period do not need an increase in oxygen to metabolize fuels because they have a lower body temperature to maintain. This perhaps represents a non-thermogenesis response to a mild cold stress within a short period of time. It is understood that change in ambient temperature effects chick body temperature, therefore understanding thermogenesis in the chick to be an innate or environmentally controlled response can perhaps lead to a better growing more efficient bird.

Glycogen averaged 4.98 mg glycogen per g wet tissue, which is similar to previous findings in hatched chicks (Freeman, 1969; Christensen, 1995). Gluconeogenesis was occurring as indicated by the increase in liver glycogen during Experiment 1. Gluconeogenic substrates present include lactate, glycerol and possible gluconeogenic amino acids as indicated by the increase in uric acid. An increase in uric acid is an indicator of the deamination of amino acids (Stevens, 1996). The efficacy of precursors to elevate plasma glucose

concentrations are ordered as lactate = glycerol > pyruvate > alanine > aspartate > serine (Langslow, 1978). These precursors are believed to be interacting in the bird to increase the liver glycogen stores during the 48-h fast. Romanoff (1967) reported the embryo at hatch will contain 56% protein, 32% lipid and 3% carbohydrates, which could be supplying enough energy through triglycerides in which we determined to decrease along with blood glucose.

Uric acid was the only substrate found to be significant with BT class, however numerical trends where found throughout experiment 1. Chicks with a HT had decreased levels of liver glycogen, and triglyceride and increased concentration of uric acid. Therefore it is hypothesized that chicks with an HT tended to utilize carbohydrate and protein stores the first few days of post hatch. Low body temperature chicks had increased concentration of liver glycogen and triglycerides with a decreased level of serum uric acid. Low body temperature chicks tend to utilize lipid stores predominately over carbohydrate stores. These trends create a story between the two body temperature classes in how they metabolize fuels differently. Whether these differences in metabolic activity effect subsequent performance is not known. Concentrations of energy and mineral indicators measured at hour 0 are similar to previous studies conducted by Mahagna and Nir, (1996).

Enhancing chick quality is essential in maximizing the genetic potential of the chick. Chicks undergo a variety of stressors from the time of hatch until maturity or market age. Whether chicks initial body temperature is controlled by innate factors, environmental factors or a combination of both is still unclear. The

present data shows potential relationships between body temperature and subsequent performance. Understanding chick thermogenesis, metabolism and subsequent growth will enhance the ability scientists and producers to enhance and maximize broiler performance. Gaining a better knowledge of how chicks react to different stressors will provide clues to help alleviate detrimental effects on performance.

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TABLE 1. Ingredient percentages utilized for starter ration

Ingredient	Percentage
Corn	61.0
Soy bean meal	29.0
Fat	1.9
ProPak	4.87
Limestone	0.97
Dical	1.2
Copper sulfate	0.03
Methionine	0.13
Vitamin Premix	0.05
Mineral Premix	0.05
Choline	0.05
Salt	0.38
Selenium Premix	0.0015
Salinomycin	0.05

[1] Premix contained: vitamin A 720 mg, vitamin D_3 8mg, vitamin E 10.0mg/g, vitamin B_{12} 3.5 mg/g, riboflavin 2.2 mg/g, niacin 6.6mg/g, d-pantothenic acid 7.055 mg/g, cholin176.36mg/g; menadione 0.52mg/g, and biotin 44mg/g.

[2]Premix contained: manganese 12%, zinc 8%, iron 6%, copper 10%, iodine 0.1% and calcium 18%.

TABLE 2. Experiment 1 variables measured in chicks at the hatchery (Hour 0)

Variable	Mean	High BT	Low BT
Body Temperature (F)	102.1	102.7[a]	101.5[b]
Weighatching temperature (g)	43.6	43.5[a]	43.7[a]
Total glycogen (g)	5.15	4.73[a]	5.53[a]
Glycogen (mg/g)	4.95	4.38[a]	5.48[a]
Glucose (mg/dl)	185.89	182.54[a]	189.00[a]
Triglycerides	113.62	10.46[a]	116.77[a]
B-hydroxybutyrate	20.68	19.79[a]	21.48[a]
Lactate	20.78	21.18[a]	20.44[a]
Blood, urea, nitrogen	11.65	11.93[a]	11.39[a]
Uric	7.41	8.06[a]	6.80[a]
Creatinine	0.22	0.20[a]	0.23[a]
Total Protein	2.24	2.36[a]	2.28[a]
Albumin	0.60	0.63[a]	0.58[a]
Lactate dehydrogenase	2038.12	1827.23[a]	2266.58[a]
Creatine kinase	3302.60	3305.94[a]	2498.99[a]
Alanine aminotransferase	2.72	2.80[a]	2.63[a]
Aspartate aminotransferase	248.48	216.31[a]	282.21[a]
Alkaline phosphatase	200.48	1978.75[a]	2181.67[a]
Sodium	149.48	149.85[a]	149.08[a]
Phosphorus	4.34	4.36[a]	4.33[a]
Calcium	7.81	8.07[a]	7.56[a]
Chlorine	122.74	123.92[a]	121.64[a]
Potassium	4.24	4.47[a]	4.18[a]
Magnesium	2.11	2.14[a]	2.09[a]

Means within a row with unlike superscripts differ ($P< 0.01$)

TABLE 3. Experiment 1 Body temperature class and age effects on performance variables including body temperature (BT), change in body temperature (DBT), weight and change in body weight

BT class	Hour	BT (F)	DBT	Weight (g)	DWT
H	0	102.7^{c}	-0.04^{d}	43.5	0.13
H	24	103.7^{a}	1.00^{c}	40.9	-2.45
H	48	103.5^{ab}	0.80^{c}	39.3	-4.06
L	0	101.5^{d}	0.14^{d}	43.7	-0.21
L	24	102.8^{c}	1.42^{b}	42.4	-1.53
L	48	103.2^{b}	1.82^{a}	40.1	-3.84

ANOVA	Df	Probability			
BTc[1]	1	0.0001	0.0001	NS	NS
Hour	2	0.0001	0.0001	0.0001	0.0001
BTc x Hour	2	0.01	0.01	NS	NS

Means within a column with unlike superscripts differ significantly ($P < 0.05$)

[1] BTc = Body temperature class

TABLE 4. Experiment 1 Interactions between hour and BT class (HT vs. LT) with serum glucose concentrations

BT class	Hour	Glucose (mg/dl)
H	0	182.54[c]
H	24	143.92[a]
H	48	154.13[ab]
L	0	189.00[d]
L	24	149.67[c]
L	48	106.80[b]
ANOVA		Probability
Sources	df	
BTc[1]	1	0.01
Hour	2	0.0001
BTc x Hour	2	0.01

Means within a column with unlike superscripts differ significantly ($P< 0.05$)
[1] BTc = Body temperature class

TABLE 5. Main effects of time (hr) on serum metabolite concentrations

	Time (hrs)		
Metabolic Indicator	0	24	48
Glycogen (mg/g)	4.94^{b}	5.79^{b}	7.37^{a}
Total glycogen (g)	5.13^{c}	6.65^{b}	9.08^{a}
Liver wt/bwt (%)	2.40^{f}	2.84^{e}	3.08^{d}
Serum chemistries			
Triglycerides (mg/dl)	113.62^{a}	99.07^{b}	80.23^{c}
Lactate (mg/dl)	20.77^{d}	12.53^{e}	9.68^{e}
B-hydroxybutyrate (mg/dl)	20.65^{a}	21.92^{ab}	18.47^{a}
Uric (mg/dl)	7.47^{b}	9.28^{b}	12.85^{a}
BUN (mg/dl)	11.68^{f}	16.15^{e}	20.72^{d}
Total Protein (g/dl)	2.24^{e}	3.17^{d}	3.20^{d}
Albumin (g/dl)	0.60^{e}	0.89^{d}	0.90^{d}
Creatinine (mg/dl)	0.22^{d}	0.15^{e}	0.22^{d}
Sodium (mmol/l)	149.48^{b}	151.96^{b}	155.70^{a}
Chlorine (mmol/l)	122.74^{e}	131.75^{d}	124.71^{d}
Potassium (mmol/l)	4.24^{f}	7.79^{d}	6.24^{e}
Calcium (mg/dl)	7.81^{e}	10.60^{d}	10.85^{d}
Phosphorus (mg/dl)	4.35^{e}	7.58^{d}	7.06^{d}
Magnesium (meq/l)	2.11^{e}	2.52^{d}	2.51^{d}
LDH[1] (U/l)	2041.80^{b}	2431.11^{a}	2376.31^{a}
ALT[2] (U/l)	2.70^{b}	1.79^{b}	4.41^{a}
AST[3] (U/l)	249.96^{e}	262.52^{e}	361.03^{d}
ALP[4] (U/l)	2090.08^{a}	2317.43^{a}	2278.48^{a}
CK[5] (U/l)	3304.80^{a}	3837.77^{a}	3176.31^{a}

Means within a row with unlike superscripts differ significantly ($p < 0.05$)
[1]LDH = Lactate Dehydrogenase; [2]ALT = Alanine aminotransferase; [3]AST = Aspartate aminotransferase; [4]ALP = Alkaline phosphatase; [5]CK = Creatine kinase

TABLE 6. Experiment 2 main effects of body temperature class with body temperature variables

	Body Temperature class	
Variable	High	Low
BTIN[1]	103.8[c]	102.4[d]
BTOUT[2]	102.4[a]	103.2[a]
DBT[3]	-1.46[f]	0.78[e]
DBTIN[4]	1.13[a]	1.04[a]
DBTOUT[5]	-0.33[h]	1.81[g]

[a – c] Means within a row with unlike superscripts differ significantly ($p < 0.05$)
[1]BTIN = Body temperature taken prior to placement into the chamber
[2]BTOUT = Body temperature taken at time of removal from the chamber
[3]DBT = BTOUT – BTIN
[4]DBTIN = change in BT from hour 0 – 48 hour post hatch
[5]DBTOUT = change in BT from hour 0 – 56 hour post hatch

TABLE 7. Ambient Temperature effects on body temperature variables

	Ambient Temperature	
Variable	30 F	32 F
BTIN	103.1[a]	103.2[a]
BTOUT	101.6[h]	103.9[g]
DBT	0.93[g]	-1.62[h]
DBTIN	1.01[a]	1.15[a]
DBTOUT	1.95[g]	-0.46[h]

[a-c] Means within a row with unlike superscripts differ significantly ($p < 0.05$)
[1]BTIN = Body temperature taken prior to placement into the chamber
[2]BTOUT = Body temperature taken at time of removal from the chamber
[3]DBT = BTOUT – BTIN
[4]DBTIN = change in BT from hour 0 – 48 hour post hatch
[5]DBTOUT = change in BT from hour 0 – 56 hour post hatch

TABLE 8. Experiment 3 body temperature class (HT vs. LT) effects on body temperature variables

	Body Temperature class	
Variable	High	Low
DBTIN	0.06[b]	1.28[a]
DBTOUT	2.99[d]	4.06[c]

[a – b] Means within a row with unlike superscripts differ significantly ($p < 0.01$)
[4]DBTIN = change in BT from hour 0 – 56 hour post hatch
[5]DBTOUT = change in BT from hour 0 – 168 hour post hatch

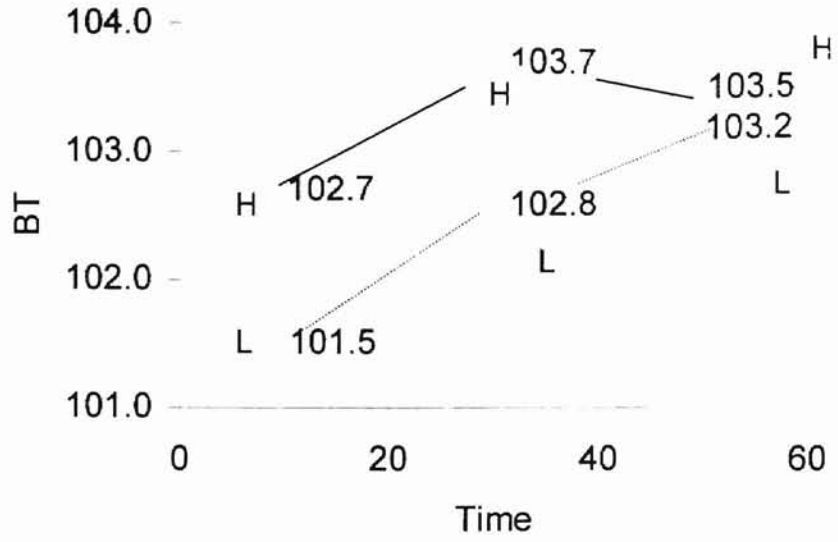

FIGURE 1. Body Temperature represented as the interaction between time and BT class

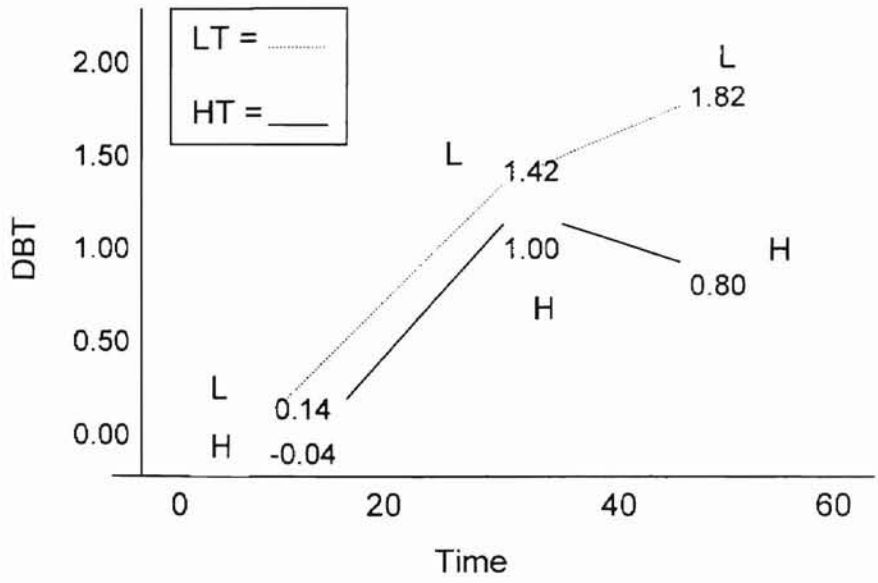

FIGURE 2. Change in body temperature (DBT) interaction between body temperature class and time

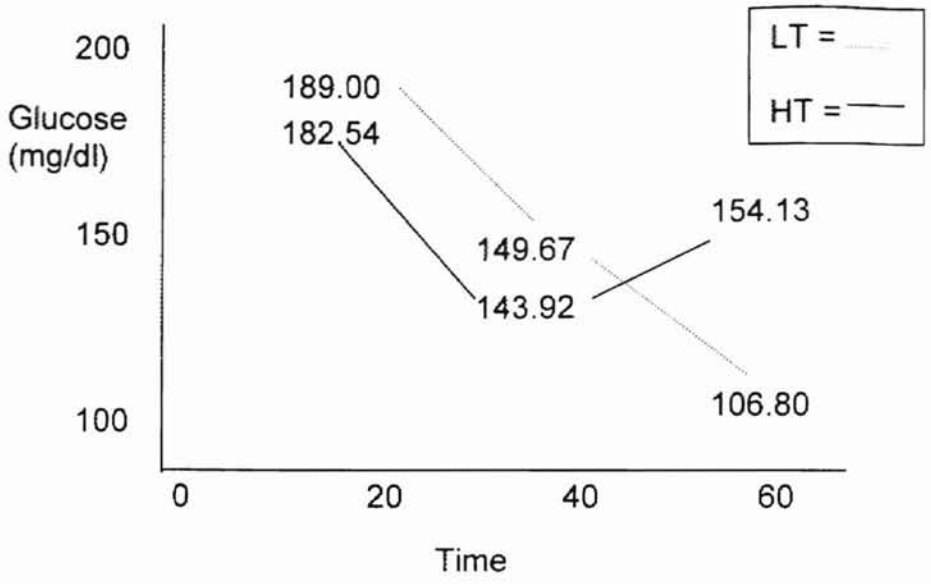

FIGURE 3 Glucose concentrations interaction of body temperature class and time

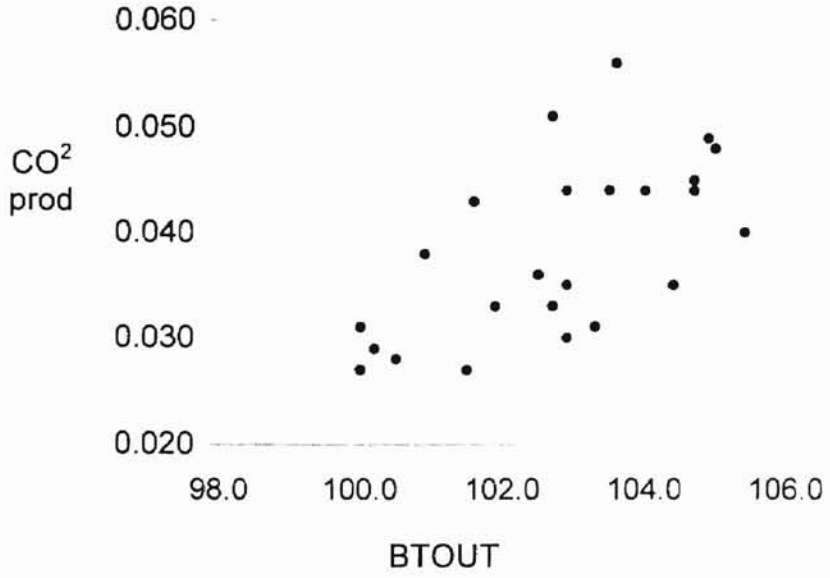

FIGURE 4. Experiment 2 relationship between carbon dioxide production and body temperatures

CHAPTER VII

INFLUENCE OF ATMOSPHERIC OXYGEN AND INITIAL CHICK BODY TEMPERATURE ON SERUM AND TISSUE CHEMISTRIES.

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Abstract: Previous studies in our laboratory suggests that initial chick body temperature (at hatch) is positively correlated with live weight gain, gain/feed ratio and ascites resistance. Three phases were conducted to evaluate effects of body temperature, at hatching, on subsequent broiler performance under varying levels of feeding and atmospheric oxygen (15.0% vs. 20.6%). Chicks were initially divided into four groups according to falling above (HT) or below (LT) the mean hatching temperature and being male (m) or female (f). Variables measured throughout the study included body temperature (BT), body weight, liver glycogen, and 16 serum analytes. Phase 1 represents data collected at the hatchery including body temperature, weight, gender, hatching time, and buggy from which chicks were hatched.

Phase 2 was conducted as a 2 x 2 x 2 factorial with two BT classes (HT vs. LT), gender (m vs. f), and atmospheric oxygen (15.0% vs. 20.6%). Variables were collected on days 0, 2, 4, and 7.

Phase 3 was conducted to examine the relationship of intial body temperature with subsequent performance utilizing 108 male Cobb broilers exposed to normal atmospheric oxygen reared to 4 weeks of age. Chicks were fasted at the conclusion of this experiment to measure basal metabolic rate in correlation with scanning data utilizing a x-ray densitometer (Hologic QDR™).
(*Key words*: broiler, metabolism, chick quality, body temperature, atmospheric oxygen)

INTRODUCTION

Numerous environmental stresses alter bird homeostasis, thereby adversely impacting bird ability to survive and/or convert feed nutrients into tissue. Stress for the chicks can begin at the hatchery in the incubators and continue to the broiler houses. Understanding these stressors on chick quality can provide a greater insight to chick performance. Homeostatic perturbation may be reflected in altered serum and tissue metabolite chemistries, that may in turn provide clues for new stress combating therapeutics.

Two stress types that have been reported to impact bird homeostasis include low ambient temperature (LAT) and atmospheric oxygen. Experiments have been conducted to identify metabolite changes occurring under (LAT) and hypoxia perturbation (Beker, 1995). Important metabolites may include the fuels utilized by the bird for energy or heat production. Low ambient temperature exposure and hypoxia have been documented to impact serum metabolites (Yersin 1992), blood hematocrit (Maxwell, 1995) and oxygen utilization (Freeman 1965), all of which influence bird survivability and feed conversion. Therefore the

objective of this study is to determine the relationship of innate (body temperature) and environmental (altitude) factors on chick quality and subsequent performance.

MATERIAL AND METHODS

Initial Bird Selection, Categorization and Processing

Seven hundred and sixty Cobb X Cobb chicks were randomly extracted from the day's hatch of 963 chicks and categorized by body temperature. Initial body temperature and weight was taken at the hatchery where chicks were sexed and separated accordingly by compartment from which each chick hatched. Chick body temperature was taken utilizing the Vet Temp Vt-100[1] infrared thermometer probe via the chick mouth. Chicks were transported and reared in calorimetric chambers described elsewhere (Belay, 1993). Data were analyzed by procedures of SAS designating chicks with high (HT) or low (LT) body temperature according to falling above or below the mean body temperature.

Tissue and Blood Analyses

Blood was collected on all chicks by cardiac puncture for phase two and via the wing vein for phase 3. Blood was collected in Corvac sterile tubes and allowed clotting for twenty-four hours. Blood was then centrifuged at 3000 rpm for 25 minutes. Serum was removed and frozen in polyethylene tubes until subsequent analysis. Blood was collected for lactate separately and treated with trichloracetic acid according to specifications of Roche. Blood serum or lactate

supernatant was analyzed utilizing a Cobas Mira[1] wet chemistry analyzer. Serum samples collected were analyzed for sodium, potassium, calcium, magnesium, chloride, phosphorus, glucose, triglycerides, total protein, albumin, uric acid, blood urea nitrogen, lactate, creatinine, lactate dehydrogenase, creatine kinase and thyroxine. Roche kits[2] utilized in determining serum concentrations include calcium (No. 44033), magnesium (No. 44169), chloride (No. 44029), phosphorus (No. 44031), glucose (No. 47382), triglycerides (No. 44120), total protein (No. 44903), albumin (42332), uric acid (No. 47273), blood urea nitrogen (No. 47380), creatinine (No. 47003), lactate dehydrogenase (No. 47259), creatine kinase (44402). Sodium and potassium values were assayed via sodium and potassium selective electrode module (No. 46997, 46998). Sigma reagents were utilized in measuring lactate (9826-A) and B-hydroxybutyrate (310-A).

Liver tissues were collected from the chicks immediately after blood collection and frozen in liquid nitrogen. Livers were wrapped and stored in aluminum foil in a –20 freezer. Analyses of liver for presence of glycogen were completed according to methods of Dreiling (1987).

Phase 1

The first phase of this trial was to determine hatching time, buggy from which chicks were hatched, gender on initial body temperature classification and weight of the chicks. On the morning of day twenty chicks, which pipped out were marked with a food coloring dye and classified as chicks that hatched early.

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[1] Roche Diagnostics Systems Inc., Montclair, NJ 07042-5199.
[2] Hoffman-LaRoche, Nutley, NJ 07042.

Chicks were placed back into the hatcher to allow other chicks to pip out and were classified as chicks, which hatched late. All chicks were pulled from the hatcher the following morning on day 21. Chicks utilized in this study were hatched from two hatcher buggies with 13 levels per buggy. Chicks were wing banded, sexed and placed in boxes according to gender, buggy and level from which chicks were hatched.

Phase 2

The second phase of this trial was conducted to examine the changes in initial body temperature and metabolic substrates overtime with chicks exposed to atmospheric stress conditions. Treatments were arranged as a 2 x 2 x 2 factorial design with two body temperature classes, (HT vs. LT), two genders (male vs. female) and two atmospheric concentrations (15.0% vs. 20.6%). Chicks were deprived of food and water and kept in transport boxes, housed at 30 C to represent a shipping environment for the first 24 hours. Eighteen chicks were distributed into 24 chambers in three rooms on day 1. Chicks were fasted of feed and given water for another 24 hours to represent a harsh shipping environment. Chicks were fed a complete starter ration ad libitum (Table1). Variables were collected on days 0, 2, 4, and 7 including body temperature, body weight, liver glycogen and blood via heart puncture. Chicks were fasted on days 4 and 7 for 12 hours to reduce the effect of feed on body temperature change.

Phase 3

The second experiment was conducted to determine initial body temperature on growth and feed efficiency. One hundred and eight male chicks

were initially placed into 36 metabolic chambers by body temperature class (HT vs. LT) and reared under a normal atmospheric environment with a mild cold stress (30 C) for 7 days. Broilers were reared until day 7 at which time 72 birds were distributed into 24 more chambers to provide more room for growth. Birds reared together until day 7 were kept together for the remainder of the experiment to be able to measure feed efficiency and growth appropriately. Body temperature and weight was taken on day 21 at which time the starter ration was replaced with a grower ration. Live weight was taken on fed birds at 7, 21 and 27 days of age with fasted weights taken on days 22 and 28. Body temperature was taken on all days except day 7. Broilers were fasted for 36 h prior to basal metabolic rate (BMR) determination. On 29 days of age the BMR period ended at which time birds were weighed, body temperature taken and bled via wing vein. Birds were gassed and immediately scanned utilizing the Hologic x-ray densitometer.

Data were analyzed utilizing General Linear Models of SAS (1988). Treatments were represented as a complete randomized block design.

RESULTS

Phase 1 results

Data collected at the hatchery including gender, hatch time, and buggy chicks hatched from were analyzed to determine with hatchery condition effects on body temperature and weight of the chick. The average body temperature was 103.1 F and ranged from 95.3 F to 107.3 F. Weights of the chicks ranged from 32.0g to 58.0g and averaged 46.6g. Chicks classified as having a body

temperature above the mean hatching temperature (HT) had an average temperature of 103.9 F and average weight of 46.4. The HT chicks ranged in body temperature from 103.1 F to 107.3 F and ranged in weight from 35.0g to 57.0g. The chicks classified as having a body temperature below the mean body temperature (LT) ranged in body temperature from 95.3 F to 103.0 F with an average BT of 102.0 F. Chicks with an initial lower BT ranged in weight from 32.0g to 58.0g with an average weight of 46.8g. Three probes were used to measure body temperature and found to be significant with body temperature taken therefore in all comparisons with body temperature probe was used as a covariant. Initial body temperature at the hatchery was found to be effected by the time in which the chicks pipped or hatched out of their shell, and the buggy from which the chicks hatched (Table 2). Chicks that hatched early had a higher ($P < 0.01$) body temperature than chicks that hatched late. The buggies chicks hatched from effected the body temperature of the chicks ($P < 0.05$). There were no three or four way interactions detected with variables measured against body temperature or weight. The interaction of gender and hatch time was significant with body temperature and weight (Table 3). This interaction was found to be due to an order of magnitude due to hatch time.

Phase 2 results

Chicks which, hatched early had a higher ($P < 0.05$) body temperature, lactate, magnesium and sodium serum concentration. Female chicks had higher ($P < 0.05$) hematocrit, triglyceride, lactate, total protein, and albumin than male chicks. Buggy from which chicks hatched also effected body temperature of

chicks. Weight was depressed ($P < 0.0001$) from age 0 to age 2 due to fasting then increased ($P < 0.0001$) from age 2 to age 4 and age 7. Weights of chicks were smaller than predicted weights due to fasting causing a disturbance in growth. The 16 serum chemistries analyzed were found to change overtime with different treatment factors uniquely effecting the serum concentrations. Serum chemistries quantified decreased ($P < 0.05$) overtime with a dramatic decrease ($P < 0.05$) of glycogen at age 7 (Table 4). On day 7, chicks exposed to high atmospheric oxygen (15.0%) depressed ($P < 0.0001$) growth compared to control birds reared at normal atmospheric oxygen. Yolk weight as a percentage of body weight decreased overtime with a significant decrease ($P < 0.05$) in birds exposed to hypoxic conditions at day 2. Hematocrit increased ($P < 0.05$) at days 4 and 7 in birds reared under a high altitude. Heart weight increased overtime with a significant depression ($P < 0.05$) in birds reared under a low oxygen concentration. (Table 5) On day 7 high altitude concentration exposure increased the serum concentrations of phosphorus (mg/dl) ($P < 0.001$), potassium (mmol/l) ($P < 0.01$), magnesium (meq/l) ($P < 0.05$), lactate dehydrogenase (U/l) ($P < 0.05$), and creatine kinase (U/l) ($P < 0.01$) with a depression in glucose (mg/dl) ($P < 0.001$) (Table 6).

Phase 3 results

Phase 3 of this study was conducted to determine initial body temperature effects on performance variables including body temperature, weight, feed consumption, feed conversion, serum chemistries, basal metabolic rate, and body composition. Birds increased ($P < 0.05$) weight overtime whether being fed

or fasted. Body temperature (via mouth, with infrared probe) decreased ($P < 0.05$) in fasted birds compared to fed birds. Feed consumption increased ($P < 0.05$) overtime while feed efficiency decreased ($P < 0.05$). (Table 7) Body temperature (via cloaca, digital probe) was taken on day 29 after a 36 hour fast. Relationships were detected between body temperature and body composition variables (Table 8). Total weight and lean increased as body temperature decreased. Basal metabolic rate increased with an increase in weight and lean mass (Table 9). There were no effect of gender, hatch or body temperature class differences detected with performance or body composition variables.

DISCUSSION

Chicks are genetically created to produce maximum profitability however chick stresses at the hatchery can neutralize this progress and have a detrimental effect on chick quality. These results indicate hatching time, buggy and incubator temperature effects initial body temperature at the hatchery. The different in male and female chicks have been studied extensively. Results reported herein indicate gender differences with serum chemistries and show physiological differences when exposed to hypoxic conditions. These factors at the hatchery can cause metabolic and performance changes in the bird later in life.

Ascites is caused by pulmonary hypertension as the result of an oxygen insufficiency to fast growing tissues. There is a linear relationship between hematocrit and heart weight which indicates an adaptation of the heart to increase its workload associated with changes in flow resistance (Yahav, 1997).

These results show chicks exposed to hypoxic conditions increased hematocrit as early as age 4 and continued to day 7 where heart weight was observed to increase. These observations indicate chicks begin adapting to hypoxic environments within 4 days of exposure. Yersin (1992) reported chicks with ascites at 7 days of age. Lactate dehydrogenase and creatine kinase increased in chicks exposed to ascitic environment as reported previously (Maxwell, 1995; Beker, 1995).

Chick body temperature gradually increases with age from 96.8 F to 104 F (Dunnington, 1984). Chick body temperature ranged from 102.0 to 103.5 in chicks while adult broiler body temperature was found to average 105.5 during days 21 to 29 which is comparable to body temperatures reported by Yahav (1997) in 8-week old broilers. Broilers that were fed had a higher body temperature compared to fasted body temperature birds as Zhou (1997) reported chicks increase abdominal temperature with food intake. Chicks with a higher lean mass and body weight had an increased basal metabolic rate. Hocking (1985) reported heavy meat type Leghorns had a lower body temperature than medium meat type broilers. A relationship was seen in broilers with heavier body weights and lean mass with lower body temperatures taken after a 36 hour fast.

Hatchery conditions effect chick quality and effects performance in chicks during the first few days after hatch. Hatchery managers must continue to provide the most efficient processing environment to enable the chick potential for growth. These results imply the effect of body temperature on weight and lean mass. Thermogenesis of the chick and broiler must continue to be studied

in order to define these changes in depth and to further understand the effect of initial body temperature on subsequent growth and performance.

Chicks are exposed to stressors before hatch and continue till optimal growth is obtained. Management practices must be followed in order to maximize bird capability. Previous research indicates the effect of hen, handling, ambient temperature, nutrient supply effect the chick and can have detrimental effects of the chicks performance if not handled properly.

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TABLE 1 Starter and grower ration composition

Ingredient	Percentage	
	Starter	Grower
Corn	61.0	65
Fat	1.91	2.87
Soybean meal	29	26
Limestone	0.9735	1.48
Dical	1.2	1.0
Copper sulfate	0.03	0.03
Methionine	0.13	0.17
Vitamin Premix	0.05	0.05
Mineral Premix	0.05	0.05
ProPak	4.87	2.53
Cholin	0.05	0.04
Salt	0.38	0.30
Selenium Premix	0.0015	0.0015
Salinomycin	0.05	0.05

[1] Premix contained: vitamin A 720 mg, vitamin D_3 8mg, vitamin E 10.0mg/g, vitamin B_{12} 3.5 mg/g, riboflavin 2.2 mg/g, niacin 6.6mg/g, d-pantothenic acid 7.055 mg/g, cholin176.36mg/g; menadione 0.52mg/g, and biotin 44mg/g.

[2]Premix contained: manganese 12%, zinc 8%, iron 6%, copper 10%, iodine 0.1% and calcium 18%.

TABLE 2 Phase 1 Hatchery condition effects on chick body temperature (BT) and live weight (WT)

	BT (F)	MSE	P-value	Weight (g)	MSE	P-value
BT class[1]						
High	103.9	0.88	0.0001	46.42	4.19	NS
Low	102.0			46.80		
Gender						
Male	103.1	1.27	NS	46.8	4.18	NS
Female	103.1			46.3		
Hatch						
Early	103.5	1.27	0.01	46.3	4.12	0.0001
Late	103.0			46.8		
Buggy						
1	102.6	1.19	0.0001	46.52	4.19	NS
2	103.5			46.63		

[1]BT class = Body temperature class

TABLE 3 Phase 1 Gender and Hatch effects on chick body temperature (BT) and live weight (WT).

Gender	Hatch	BT[1] (F)	Weight (g)
F	E	103.2[b]	45.1[bc]
M	E	103.8[a]	43.6[c]
f	L	103.1[b]	46.4[b]
M	L	103.0[b]	47.1[a]
Mean		103.1	46.59
MSE		1.26	4.11
Anova		df	
Probe[2]	1	0.05	*
Hatch	1	0.01	0.0001
Gender	1	NS	NS
G*H[3]	1	0.05	0.05

[1] BT = Body temperature, [2] Thermometer probe utilized to estimate body temperature was used as a covariant. [3]G x H = gender x hatch,
* note probe was not used as a covariant with weight

Table 4 Phase 2 Effects of age on performance and serum chemistry variables

Variables	Days of Age 0	2	4	7	mean	MSE	P-value
Weight (g)	47.1[c]	41.6[d]	62.2[b]	100.2[a]	70.15	3.63	0.0001
Body Temperature (F)	103.0	102.9	102.9	102.9	102.94	1.25	NS
Yolk wt %bwt	8.62[a]	6.45[b]	1.33[c]	0.38[d]	3.29	2.08	0.0001
Glycogen (mg/g)	*	7.16[b]	10.62[a]	1.90[b]			0.001
Glucose (mg/dl)	121.5[c]	148.5[b]	179.4[a]	154.3[b]	158.91	26.6	0.0001
Triglycerides (mg/dl)	125.1[a]	112.0[b]	88.7[c]	74.3[d]	93.07	23.0	0.0001
Lactate (mg/dl)	16.45	16.45	16.45	17.36	16.59	4.37	NS
Uric Acid (mg/dl)	11.56[a]	10.53[a]	4.10[b]	3.94[b]	6.44	3.9	0.0001
BUN[1] (mg/dl)	18.18[b]	23.38[a]	2.00[c]	2.06[c]	9.29	2.4	0.0001
BHA[2] (mg/dl)	10.14[a]	11.12[a]	5.89[b]	12.10[a]	9.64	4.3	0.0001
Total Protein (g/dl)	3.25[b]	3.52[a]	2.75[c]	2.74[c]	3.00	0.32	0.01
Albumin (g/dl)	1.14[ab]	1.20[a]	0.94[c]	1.06[b]	1.07	0.17	0.0001
Creatine (mg/dl)	0.21[a]	0.15[b]	0.12[c]	.012[c]	0.13	0.06	0.01
Chloride (mmol/l)	110.14[ab]	113.98[a]	102.65[c]	105.32[b]	106.42	8.75	0.05
Magnesium (meq/l)	2.13[b]	2.28[a]	2.00[c]	2.18[b]	2.15	0.27	0.05
Calcium (mg/dl)	7.43[a]	7.91[a]	5.66[b]	5.25[c]	6.22	1.13	0.01
Phosphorus (mg/dl)	8.07[a]	6.57[c]	5.71[d]	7.15[b]	6.55	1.11	0.01
Sodium (mmol/dl)	160.24[b]	165.89[a]	143.32[d]	144.65[c]	150.92	4.51	0.05
Potassium (mmol/l)	8.81[a]	7.23[c]	7.89[b]	9.13[a]	8.14	1.22	0.01
LD[3] (U/l)	2256.82[a]	1987.46[b]	1710.62[c]	2052.75[ab]	1932.4	440.59	0.05
CK[4] (U/l)	3194.0[a]	2556.9[b]	1589.1[c]	2805.1[a]	2341.7	781.11	0.05

[1] BUN = Blood, urea, nitrogen, [2] BHA = B-hydroxybutyrate, [3]LD = Lactate Dehydrogenase, [4] CK = Creatine Kinase

Table 5 Phase 2 Day and oxygen effects on physiological variables

Day	Oxygen (%)	Weight	Yolk [1]	HMT[2]	Heart[1]	AHI[3]
2	15.0	41.8^{d}	5.66^{b}	32.7^{a}	0.86^{c}	
2	20.6	41.5^{d}	7.19^{a}	32.1^{a}	0.87^{c}	
4	15.0	62.0^{c}	1.43^{c}	28.1^{c}	0.89^{c}	0.21
4	20.6	62.6^{c}	1.12^{cd}	24.9^{d}	0.87^{c}	0.20
7	15.0	97.5^{b}	0.33^{d}	29.5^{b}	1.04^{a}	0.26
7	20.6	102.9^{a}	0.42^{d}	25.8^{d}	0.93^{b}	0.26
Mean		73.36	2.78	28.95	0.64	0.24
MSE		3.44	1.96	2.97	0.08	0.15
				Probability		
Day	2	0.0001	0.0001	0.0001	0.0001	0.01
Oxygen	1	0.0001	0.10	0.0001	0.001	NS
D x O[4]	2	0.0001	0.05	0.01	0.0001	NS

[1] Yolk and heart is presented as a percentage of body weight, [2] HMT = Hematocrit, [3] AHI = Ascites Heart Index,
[4] D x O = Day x Oxygen

Table 6 Phase 2 Day and oxygen effects on serum chemistries

Day	Oxygen	Glucose (mg/dl)	Phosphorus (mg/dl)	Potassium (mmol/l)	Magnesium (meq/l)	LD[1] (U/l)	CK[2] (U/l)
2	15.0	147.5^{b}	6.51^{b}	7.21^{d}	2.29^{a}	1923.3^{b}	2501.6^{b}
2	20.6	149.6^{b}	6.62^{b}	7.25^{d}	2.28^{a}	2047.1^{ab}	2608.4^{b}
4	15.0	177.5^{a}	5.77^{c}	8.00^{c}	2.03^{bc}	1724.1^{c}	1568.9^{c}
4	20.6	183.5^{a}	5.58^{c}	7.67^{cd}	1.94^{c}	1685.1^{c}	1628.6^{c}
7	15.0	140.8^{b}	7.69^{a}	9.65^{a}	2.28^{a}	2140.2^{a}	3091.8^{a}
7	20.6	168.1^{c}	6.63^{b}	8.64^{b}	2.09^{b}	1969.8^{b}	2518.5^{b}
Mean		160.46	6.49	8.11	2.15	1915.9	2301.3
MSE		25.4	1.07	1.17	0.26	438.53	766.77
Day	2	0.0001	0.001	0.0001	0.0001	0.0001	0.0001
Oxygen	1	0.0001	0.001	0.001	0.01	NS	NS
D x O[3]	2	0.001	0.001	0.01	0.05	0.05	0.01

[1] LD = Lactate Dehydrogenase, [2] CK = Creatine Kinase, [3] D x O = Day x Oxygen

Table 7 Phase 3 Effects of fed and fasted broilers on performance variables

	Fed periods (age in days)			Fasted periods (age in days)			
	7	21	27	22	28	mean	MSE
Weight	193.9[e]	912.5[c]	1489.7[a]	856.0[d]	1408.3[b]	942.13	100.48
BT		106.0[a]	105.7[b]	105.5[bc]	105.3[c]	105.5	0.85
Fdcons	161.0[c]	1092.11[b]	2003.16[a]	*	*	1079.7	101.6
Fd/gain	0.83[c]	1.22[b]	1.37[a]	*	*	1.34	0.12

[a-e] unlike superscripts within a row differ ($P < 0.05$)

Table 8 Phase 3 Body temperature effects on body composition variables

		BT[1] 106.1 (F)			
		Std dev	R	P - value	N
Total (g)	1280.1	110.2	-0.38695	0.05	40
Fat (g)	156.4	32.1	-0.07708	NS	40
Lean (g)	1104.1	101.3	-0.39381	0.05	40

[1] BT = Body temperature

Table 9 Phase 3 Basal metabolic rate on body composition variables

		BMR[1] 24.56			
		Std dev	R	P - value	N
Total (g)	1251.0	78.0	0.98130	0.0001	40
Fat (g)	155.6	34.0	0.18499	NS	40
Lean (g)	1076.3	76.1	0.91321	0.0001	40

[1] BMR = Basal metabolic rate

CHAPTER VIII

SUMMARY AND CONCLUSIONS

Numerous studies conducted indicate the stressors upon which chicks are exposed and must deal with on a daily basis. The conditions can be defined as innate or environmental factors. Geneticists today have produced a fast growing feed efficient bird. However, indirect characteristics have been exposed such as increased leg problems. It has been hypothesized that birds are changing hormonal, thermogenic, and physiological attributes in order to maximize optimum production.

The nutritionists' goal in turn is to supply broilers with the nutrients to enable them to reach maximum growth and conversion efficiency without minimum stress to the bird. However, stressors including change in ambient temperature, low atmospheric oxygen, improper nutrition, diseases and poor hatchery condition exposure to these birds is at times inevitable. Understanding the birds metabolic, physiological and performance characteristics under these stressors can lead to therapeutic options to help alleviate these birds.

Heat stress and coccidia decrease weight, feed efficiency and increases mortality. The first study was conducted to quantify Betaine effects on bird thermobalance (heat production, evaporative cooling, body temperature, water balance), and urine production while exposed to heat stress with or without cocci challenge. Phase 1 of this study betaine supplementation of 0.15% improved

feed efficiency in chicks at 15 days of age. Chicks' exposed to cocci challenge during phase 2 of this study reduced all performance variables including live weight, feed efficiency, water consumption and water to feed ratio. Betaine was fortified in the drinking water at 0.10% during phase 3, which represented the thermobalance phase of this study. Chicks exposed to high ambient temperature reduced all performance variables including weight, feed efficiency, with an increased water consumption, urine production, and body temperature. These data indicate the extreme detrimental effects of heat stress on water retention of broilers. Birds with cocci history showed increased urine production, body temperature and evaporative cooling independent of heat stress exposure. Betaine improved water retention by reducing urine production. Chicks with cocci challenge increased water consumption 780% during cycling heat stress while birds with cocci challenge supplemented with betaine with high ambient temperature exposure increased water consumption only 47%. This study indicates betaine fortification improves water balance of birds while under heat stress exposure. It is hypothesized that betaine may be used as a therapeutic additive to birds exposed to high ambient temperature exposure.

The objective of the second study was to determine pantothenic dietary levels and therapeutic effects in birds reared under conditions predisposing to ascites. Pantothenic acid deficiency in the bird has been reported to decrease gain and feed efficiency, increase dermatitis, and increase the heat increment of feed possibly increasing the susceptibility of ascites. Birds reared under conditions predisposing to ascites had increased AHI and hematocrit. In this

study Pantothenic acid did not show to have efficacy on reducing the onset of ascites.

There are many procedures in estimating body composition of the chicken however tend to be laborious and costly to accomplish. Birds utilized to determine whole body composition are often sacrificed making it difficult for the researcher to follow the true composition of a single bird throughout its' growth period. The objective of the third study was to determine the efficacy and accuracy of utilizing the Hologic x-ray densitometer with chicks exposed to a hypoxic environment. Protein, ash and fat estimated for x-ray densitometry were regressed against protein, fat and ash estimated by proximate analysis to determine the efficacy of utilizing the x-ray densitometer as a means of following body composition changes overtime. Significant regression equations were found when predicting body composition at day 14 yet not on day 0. Therefore we conclude the x-ray densitometer has potential to be utilized as a way to determine body composition in older broilers utilizing appropriate regression equations.

The fourth study was conducted to determine initial body temperature effects on chick performance. The objective of the study described herein is to determine if chicks differing in body temperature set point also differ in growth rate, liver glycogen and serum chemistries. Phase 1 of this study chicks were classified as having a high or low body temperature whether falling above or below the mean body temperature taken at the time of hatching. Body temperature, live weight, 19 serum chemistries, and liver glycogen were

measured in chicks on 0, 2, 4, and 7 days of age. Chicks increased body temperature overtime whether classified as having a high or low body temperature. The low body temperature chicks increased their body temperature at a greater rate than high body temperature chicks. The defined body temperature classes tended to uniquely metabolize substrates differently. In phase 2 of this study chicks were exposed to a mild cold stress for 24 hours. Low ambient temperature reduced all body temperatures however those chicks designated as high body temperature chicks reduced their body temperatures to a greater extent. Body temperature class (high vs. low) and time of processing (0, 24, or 48 h) effected serum glucose concentration. Serum glucose decreased due to fasting however chicks with a low body temperature decreased glucose concentration the greatest. This study indicates body temperature of chicks could be an innate characteristic and may have effect on subsequent performance.

The fifth study was conducted to determine initial body temperature effects on growth, feed efficiency, basal metabolic rate and body composition variables. One hundred and eight chicks were reared to 4 weeks of age. Chicks were designated as having a high or low body temperature at the hatchery by falling above or below the mean body temperature at hatch. Chicks, which hatched early, were held in the hatcher 24 hours before removal to allow those chicks, which had not pipped to hatch. Chicks that hatched early had a higher body temperature and lower weight than chicks that hatched 24 hours later. Body temperature and weight were taken throughout the study with serum chemistries

and body composition variables taken at the conclusion of the study. Broilers, which were fed, had a higher body temperature than fasted birds. At day 29 chicks with a lower body temperature had higher live weights and lean mass. Birds with a higher basal metabolic rate had higher body weights and lean mass. These results describe hatchery condition effects on the chick that may lead to metabolic and or physiological changes in subsequent performance. These results indicate the effect of feeding on body temperature. These data exhibit potential implications to the effects of thermogenesis capability on subsequent performance.

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